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OF THE

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Royal Army Medical Corps

EDITED BY

COLONEL W. H. HORROCKS, K.H.S.

ISSUED MONTHLY

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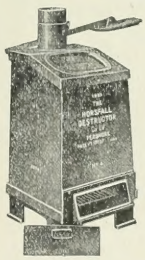

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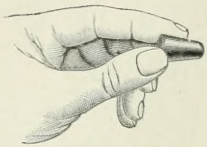

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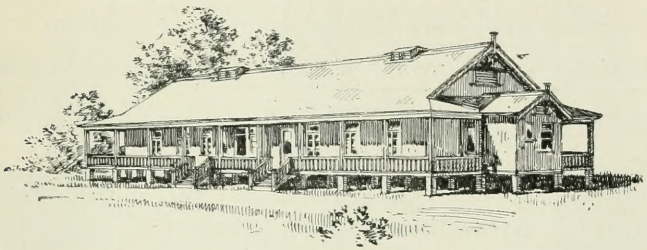

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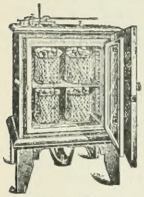

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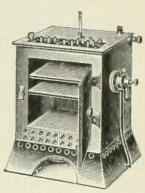

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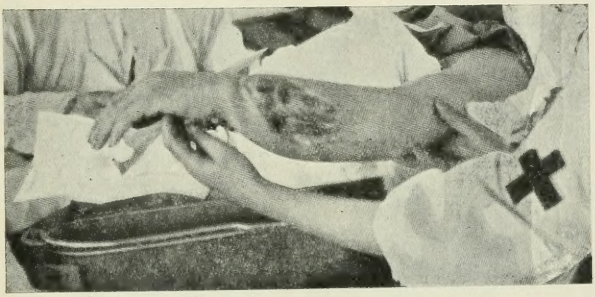

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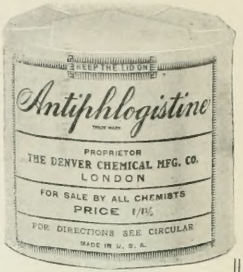

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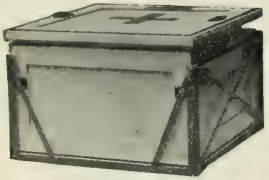

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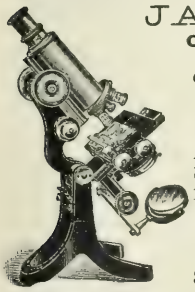

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To face first matter

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Original Communications.

REPORT ON THE RESULTS OF THE BILHARZIA MISSION IN EGYPT, 1915.

BY TEMPORARY LIEUTENANT-COLONEL ROBERT T. LEIPER, D.Sc., M.B.
Royal Army Medical Corps.
Helminthologist to the London School of Tropical Medicine.

PART I: TRANSMISSION.

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INTRODUCTORY.

THE lesions of bilharziosis are mainly due to the damage caused by the hard-shelled eggs of the parasite acting as foreign bodies in the tissues. Once the eggs are laid it is impossible to destroy them by treatment owing to the chitinous nature of the egg-shell. The effects continue with ever-increasing risk of complications and sequelæ for a number of years; until, in fact, the eggs succeed in escaping from the organ, bladder or bowel, in which they are deposited.

The only mode of dealing successfully with bilharziosis is to prevent its spread to uninfected persons.

The great economic loss resulting from widespread bilharzial infection was commented upon by Lord Kitchener in his annual report on Egypt for 1913, and the view is therein expressed that "it is high time that serious steps should be taken to prevent the continuity of infection that has been going on so long in this country."

The intractable character of the disease and the corresponding financial burden can be graphically illustrated from the experience of the Army. During the Boer War six hundred and twenty-five men were infected with bilharziosis in South Africa. In 1911, three hundred and fifty-nine of these were still on the list, exclusive of those meanwhile permanently pensioned. The cost to the State for "conditional" pensions for these three hundred and fifty-nine men was about £6,400 per annum. The "permanent pensions" already allotted amounted to an additional sum annually of £4,400 [128].

In the Nile Delta, bilharziosis is much more widespread and more severe in its manifestations than in South Africa. With the concentration of troops in Egypt it became desirable that the preventive measures taken against this disease should be made with a clear appreciation of the factors and the conditions under which

the disease is contracted and propagated. Unfortunately, although the parasitic nature of the disease had been established in 1851 nothing definite had yet been discovered of the life-cycle of the parasite or its manner of entering the human body. Early writers had supposed that the bilharzia worm, like other trematodes, required to undergo a metamorphosis in a molluscan intermediary before it became capable of infecting another person. In 1894, however, Looss formulated the hypothesis that the disease is communicable directly from man to man. In submission to his great authority in helminthological matters and his skill in dialectics, practically all research on the transmission of African bilharziosis undertaken during the last twenty years has been directed to the experimental verification of this hypothesis. During 1914 the author was in charge of the first Wandsworth Expedition of the London School of Tropical Medicine investigating the mode of spread of trematode infections of man in the Far East. Results which threw discredit upon the Looss hypothesis had just been acquired when the outbreak of war rendered field work impossible and necessitated an early return. The new facts were communicated to the War Office, and approval was given for the author to proceed to Egypt "to investigate bilharzia disease in that country and advise as to the preventive measures to be adopted in connection with the troops." Drs. R. P. Cockin and J. G. Thomson were associated with the author in the inquiry, and Private W. McDonald was transferred from the Sixth Essex (T.), as laboratory assistant.

The Committee of the London School of Tropical Medicine granted permission to all three members of their staff, while retaining their appointments, to accept temporary commissions for the period of the investigations. The requisite scientific apparatus was furnished by the School Committee. The Medical Research Committee (Insurance Act) allocated a special fund for all necessary field and other expenses incidental to the research. The Mission arrived in Egypt on February 8 and left again on July 15.

Historical.

Evidence of the occurrence of bilharzial disease in Egypt in ancient times has been found in early Egyptian records [388] and in the bodies of mummies now in the Cairo Museum [416]. In his Memoirs, Larrey [264] notes that symptoms of the disease were

frequent among the French troops during the Napoleonic Invasion of Egypt, 1799-1801. It was not until 1851, however, that the cause was recognized. In that year, Dr. Th. Bilharz, Assistant Professor at the Medical School in Cairo, wrote several letters to von Siebold announcing the discovery of a bi-sexual distome, which he named *Distoma hæmatobium*, and successfully establishing a definite relationship between this trematode worm and the symptoms of dysentery and hæmaturia resulting from the corresponding lesions of the intestine and bladder. These letters were published by von Siebold in the *Zeitschr. f. wissenschaft. Zool.* in the form of three articles during 1852 and 1853 [25], [26], [27].

Discovery.

The disease occurs in most parts of Africa, in the outlying islands of Cyprus, Madagascar, Mauritius and Réunion; in Mesopotamia, West Indies, Porto Rico and Martinique especially, and in South America. Cases have occurred sporadically also in India, Australia, and England. The accompanying map gives the regions from which cases have actually been reported, with reference numbers to the papers in the Bibliography.

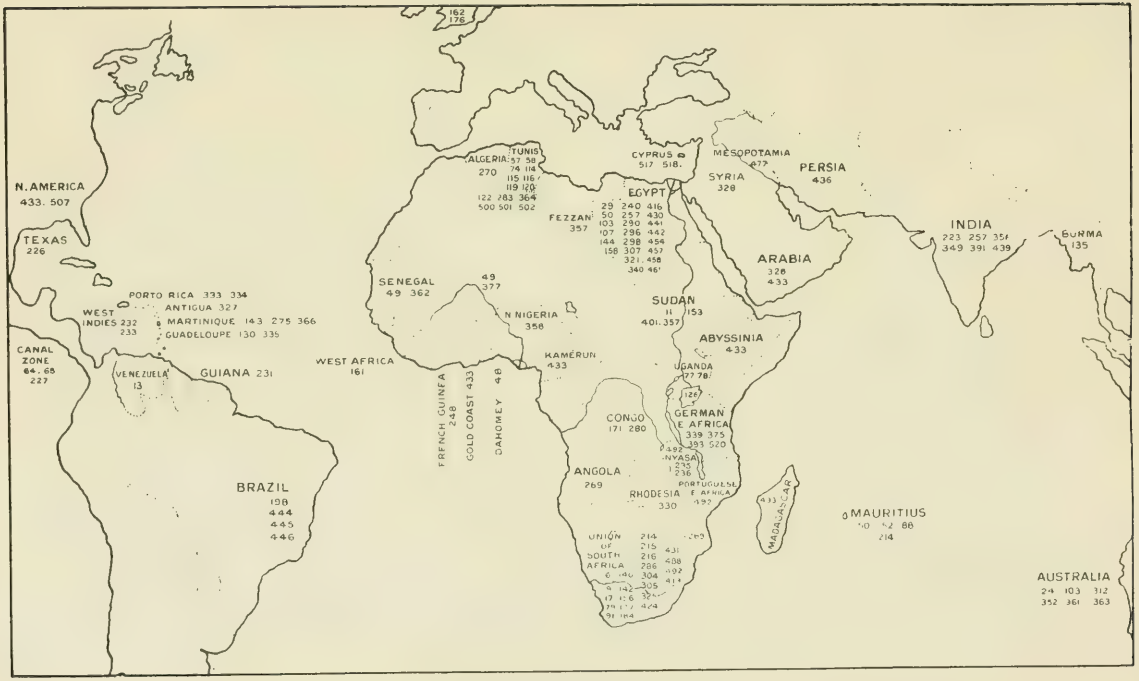
Geographical Distribution.

In 1858 Weinland [515] created a special genus *Schistosoma*, for the *Distomum hæmatobium*. In the following year Cobbold [92] discovered a similar parasite in the mesenteric veins of the sooty mangaby, *Cercocebus fulginosus*. For this he used the new generic and specific terms, *Bilharzia magna*. Later, Cobbold accepted Leuckart's view that this species was identical with that described by Bilharz.

Nomenclature.

In 1864 Harley [214] showed that the endemic hæmaturia, common in certain parts of the Cape of Good Hope and Natal was due also to a species of Bilharzia, which he named *B. capensis*. Writing in 1871, Harley [216] confessed, "I have never had much doubt of the identity of the North and South African parasite, still, I can only deal with facts, and my position with regard to the question is pretty much the same as it was seven years ago. . . ." "Both Bilharz and Griesinger describe and figure *two* forms of egg, the one with a terminal and the other with a lateral spine. In all my own cases I can say positively that only one form of egg has existed, viz., that with a terminal spine. I have never seen any egg with even a tendency to the formation of a side spine." This differentiation of species was strongly opposed by Cobbold.

It would thus appear that the name *B. hæmatobia* was *primarily* associated by Bilharz with the parasites from the mesenteric veins



Distribution of Bilharzia Hæmatobia.

Key Map to Bibliography.

and those consequently concerned in the production of intestinal lesions, while the name *B. capensis* was proposed by Harley for that specially inducing vesical lesions.

In 1893 Manson [328] suggested, on grounds of dissimilar geographical distribution, that the vesical and intestinal forms of the disease were of separate origin. Adopting this suggestion, Sambon [425], in 1907, formally created a new species, *Schistosomum mansoni*, for the lateral-spined egg. Its validity was bitterly criticized by Looss [295], and the exact relation of this new name to the older specific names, *B. hæmatobia*, *B. capensis*, and *B. magna*, still remains to be finally established.

Terminology. The disease has been variously named: (*a*) After the discoverer, bilharzia disease, bilharziosis, bilharziasis; (*b*) after the generic name of the parasite, schistosomiasis; (*c*) after the chief clinical manifestations, e.g., endemic hæmaturia, bilharzial dysentery, verminous cystitis, Cape hæmaturia, Egyptian hæmaturia; and (*d*) after the pathological situations, e.g., rectal bilharziosis, hepatic bilharziosis.

Search for Intermediary. Bilharz does not appear to have made any observations upon the life-cycle or the probable mode of spread of the disease.

In 1854 Griesinger [200] conjectured that the young of Bilharzia "existed in the waters of the Nile, in the fishes which therein abound, or even in bread, grain, and fruit."

Harley [214], in 1864, wrote "according to the observations of Professor Siebold on the trematode worms, it may safely be assumed that between the ciliated embryo above described and the adult sexual animal there are probably two other distinct forms which serve to complete the chain of metamorphosis connecting these two extremes of development. What these forms are, and what their transmigrations, are questions which require careful elucidation. The ciliated embryo is adapted for an aquatic existence. Swimming freely about, these minute organisms probably come in contact with certain mollusca and become developed within them into what have been called cercaria sacs."

In his text-book on "Entozoa," published in 1864, Cobbold [95], after quoting Griesinger, remarks, "I think it is more probable that the larvæ, in the form of cercariæ, rediæ and sporocysts, will be found in certain Gastropod molluscs proper to the localities from whence the adult forms have been obtained."

The credit of having made the first attempts to trace the life-cycle through an invertebrate intermediary must be given to

Cobbold. His experiments, begun in 1870, were made in England with eggs obtained from persons who had returned from South Africa with hæmaturia. These experiments, which proved negative, are recorded in an article [101] "On the Development of *Bilharzia hæmatobia*," in 1872, in the following words:—

"I naturally sought for the possible intermediary bearers of Bilharzia among fresh-water molluscs and small crustacea. . . . I tried to induce the ciliated embryos to enter into the bodies of a great variety of animals such as gammari, dipterous larvæ, entomostraca, limnæi, paludinæ, different species of planorbis, and other fresh-water molluscs, but neither in them nor in sticklebacks, roach, gudgeon, or carp, did they seem inclined to take up their residence. These experiments, however, are by no means conclusive, since the conditions under which the experiments were made departed in several respects from those that are presumably essential to success in the ordinary course of Nature."

The first, and in many respects the most sustained effort to elucidate the problem in an endemic area was made by the Italian helminthologist Sonsino, principally during the last two years of his stay in Egypt from 1874 to 1885.

The report of his "Ricerche sullo sviluppo della Bilharzia Hæmatobia," issued in 1884 [462], shows that Sonsino's investigation followed upon rightly conceived lines. Having accepted Chatin's view that the presence of germinal cells in the ciliated embryo indicated preparations for a metamorphosis as in other trematodes, he attempted, in the endemic area:—

(*a*) To infect, experimentally, mollusca kept in an aquarium.

(*b*) To find larval Bilharzia naturally infecting some species of mollusc or insect.

The following species are definitely listed as giving negative results—(*a*) to experimental infection: **Vivipara unicolor, Cleopatra cyclostomoides, Cleopatra bulimoides, Physa Alexandrina,** and **Melania tuberculata,** and (*b*) to dissection: **Vivipara unicolor, Cleopatra cyclostomoides, Cleopatra bulimoides, Physa Alexandrina, Melania tuberculata, Physa micropleura, Physa Innesi, Limnæa natalensis, Planorbis schweinfurthi, Unio campanii, Unio Jickeli, Corbicula spp.,** and **Spatha caillaudi.**

The dissections revealed, however, a large number of trematode larvæ in the molluscs of Egypt. These developmental forms were described in a notable communication in 1892 [472].

From these investigations Sonsino concluded that "non é improbabile che la bilhariza che per tante particolaritá s'allontana

dagli altri generi di distomi offra ancora questa singolarità di avere per ospiti intermediarii animali di classi diverse da quelle che servono allo oviluppo degli altri distomi o quella di compire tutto il suo vilo vitalé in parte libero nell' acqua e in parte parasita in un solo ospite finale e forte senza reproduzione alternante."

In 1893-94, no fewer than three special missions visited North Africa to investigate Bilharzia transmission. Sonsino, then lecturer on parasites in the University of Pisa, proceeded to Tunis from Italy. The French Government sent Professors Lortet and Vialleton to Egypt. The University of Leipzig provided funds for Dr. Looss, Assistant to Professor Leuckart, to proceed to Alexandria.

Sonsino [469] reported that, after many experiments with different kinds of fresh-water molluscs (including **Melania tuberculata, Melanopsis præmorsa** and ? **Amnicola similis**) and arthropods, he had succeeded in obtaining evidence that a small crustacean was an infected intermediary host of Bilharzia; that the Bilharzia had a life-history differing from the typical one of the digenetic trematodes, as represented by *Fasciola hepatica;* that it required an intermediary host and underwent a metamorphosis, without asexual (or alternation of) generation, "thus resembling the holostomes"; that the free embryo having effected an entrance proceeded to encyst itself and that the encysted larva, being transferred with the crustacean in drinking water to the human stomach, was then set at liberty. After penetrating the intestinal walls, it arrived in the portal vein, where, presumably, it completed its development.

Further work led Sonsino [471], in the following year, entirely to withdraw these conclusions as untenable. In the same article he records without comment the discovery of larvæ in **Melania tuberculata** which he refers to **Cercaria ocellata** and which, as will be shown later in this report, is closely allied to Bilharzia.

Lortet and Vialleton [303] failed to transmit the disease directly to animals by feeding and inoculation, or to obtain infection of plants, aquatic arthropods, or molluscs; the following species of mollusca being specifically mentioned: **Unio ægyptiacus, Corbicula consobrina, Physa acuta, Vivipara unicolor, Lanistes carinatus, Lanistes bolteniamus,** and **Melania tuberculata.**

Further experimental work was done by Lortet at Lyons in France, with several local species of Limnæa, but without success.

Later, in 1905, Lortet [302] published these "Expériences nouvelles sur le developpement et la mode de pénétration du Bilharzia

hæmatobium": "Dans un aquarium nous avons versé tous les jours des embryons et des œufs de *Bilharzia* qui étaient ainsi mis en contact avec diverses espèces de mollusques aquatiques appartenant aus genres Limnæa et Planorbis.

"Dans quelques-uns de ces mollusques, nous avions rencontré des kystes que nous pensions être différents de ceux de la douve commune. Des milliers de ces mollusques que nous supposions devoir être infectés on été ingéré, mêlès à du son, par un veau, des moutons, ainsi qui par un singe. Les resultats ont été également négatifs."

Dr. Looss' report of his investigation is contained in a critical article [288] upon a paper of Dr. G. S. Brock [67]. His conclusions are copied here in extenso:—

"Als das Wahrscheinlichste und zunächst zu Erwartende war es natürlich anzusehen, dass der aus der Eischale befreite Embryo nach Art der übrigen Distomenembryonen in einen Zwischenwirt aus der Klasse der Weichtiere eindringe. Ich wiederholte bei den Experimenten in dieser Richtung die Versuche Cobbold's und Sonsino's, aber mit dem gleichen, durchaus negativen Erfolge. Weder bei den häufigsten Gasteropoden des Nildeltas (**Cleopatra bulimoides, Melania tuberculata, Vivipara unicolor, Lanistes carinatus, Physa Alexandrina**) noch bef Lamellibranchiaten (**Corbicula Caillaudi**) zeigte sich irgend eine Infektion, gleichviel, ob dieselbe bei Tage, im direkten Sonnenlichte, oder bei Nacht, ob sie bei erhöhter oder gewöhnlicher Temperatur, in grossen oder kleinen Bassins versucht wurde. Gleich negativ waren die Bemühungen, in denselben Mollusken, die auf oft mehrtägigen Exkursionen an notorischen Infektionsherden des Deltas gesammelt waren, irgend eine Cercarienform aufzufinden, welche auch nur mit einiger Wahrscheinlichkeit auf die Bilharzia hätte bezogen werden können. Namentlich diese letzteren negativen Erfahrungen sind es, welche mich veranlassen, die Mollusken jetzt definitiv als Zwischenwirte für unseren Wurm ausser Rechnung zu stezen. Derselbe ist in Aegypten so häufig, viel häufiger, als es die bisher veröffentlichten Statistiken die allerdings Stadt und Landbevölkerung gleichmässig betreffen, während ich mich bei meinen Untersuchungen hauptsächlich an die letztere hielt—nachweisen, dass man, falls eine Cercarie der Bilharzia im Freien existierte, sie daselbst sicher und auch häufig finden müsste. Und das um so mehr, als die Mollusken der Nilwässer ungemein häufig Cercarien beherbergen: 50-60 Proz. zeigen sich fast überall infiziert, an manchen Orten aber erwiesen sich von 100 untersuchten nur 2 frei von Parasiten!

"Dasselbe Resultat ergaben in ganz der gleichen Weise angestellte

Versuche mit Crustaceen und Insektenlarven (Daphnia, Cyclops, Chironomus, Culex, Ceratopogon, Ephemera, u.a.).

"Nicht glücklicher verliefen, nachdem so auch Crustaceen und Insektenlarven als mutmassliche Zwischenträger des Wurmes hatten von der Liste gestrichen werden müssen, entsprechende Versuche mit kleinen Würmern und Fischen."

"Das eben betonte Verhalten der Embryonen anderen Tieren gegenüber war es nun auch wesentlich, welches mich schliesslich zu der definitiven Ueberzeugung brachte dass die Uebertragung der Embryonen mit Hilfe eines Zwischenträgers aus der Klasse der niederen Tiere nicht vor sich gehen könne. Es blieb deshalb nur noch die Möglichkeit übrig, dass der Embryo direkt in den Menschen gelange und dort zu einer Sporosyste auswachse, die ihre Brut dann an ihren Träger abgebe."

Naturally, the most likely course and that which one expected *a priori* was for the embryo after escaping from the egg-case to penetrate, in a manner similar to that observed in other species of distoma, into some intermediate host appertaining to the class of mollusca. In my experiments in this connexion I repeated those of Cobbold and Sonsino, but with the like absolutely negative results. Neither in the most frequently occurring gastropods in the Nile Delta (*Cleopatra bulimoides*, *Melania tuberculata*, *Vivipara unicolor*, *Lanistes carinatus*, *Physa Alexandrina*) nor in the lamellibranchiates (*Corbicula Caillaudi*) was any sign of infection to be detected, whether the experiment was carried out in the day-time, in direct sunlight, or at night, whether at high temperatures or normal ones, whether in large or in small troughs. Equally negative were the results of attempts to find cercaria forms, which could with any probability be referred to *Bilharzia*, in samples of the same mollusca collected on excursions, sometimes extending over several days, from notorious foci of infection in the Delta. It is more particularly these latter negative experiences that induce me now definitely to exclude mollusca as the intermediate hosts of our parasite. The latter is of such frequent occurrence in Egypt, far more so than would appear from the published statistics—though I admit that these refer to both urban and rural populations, whereas my experiments refer mainly to the latter—that the cercaria form of the Bilharzia, if such exists in a free state, ought to be met with frequently and with certainty under such conditions. And this should be so all the more, considering that the mollusca found in the waters of the Nile harbour cercariæ with remarkable frequency; nearly everywhere from fifty to sixty per cent. are found to be infected, and in some localities only two out of every hundred proved to be free from parasites.

Experiments conducted in a similar manner with crustacea and insect larvæ (daphnia, cyclops, chironomus, culex, ceratopogon, and others) yielded the same result.

Crustacea and insect larvæ having had to be struck off the list of likely hosts of Bilharzia, analogous experiments were carried out with small worms and fish; the results were just as unsatisfactory.

The above described behaviour of the embryos towards other animals was in the main what led me finally to the definite conviction that probably no transference of the embryos by means of an intermediate host appertaining to the classes of the lower animals takes place. Hence the only possible solution that remained was that the embryo reaches man directly, and develops into a sporocyst in the human host, the offspring of the cyst being subsequently distributed to its host.

In 1896 Looss published, in "Recherches sur la Faune parasitaire de l'Egypte" [290], a detailed account of the larval forms

met with in the course of his dissections. With five exceptions these had already been described, in less detail, by Sonsino in 1895. It is noteworthy that seven of the forms described by Sonsino do not appear to have been found by Looss. The molluscs from which the larval forms were obtained belonged to the following species: **Vivipara unicolor, Cleopatra cyclostomoides, Cleopatra bulimoides, Physa alexandrina, Physa micropleura, Melania tuberculata, Limnæa natalensis,** and **Corbicula Caillaudi,** all of which had already been examined by Sonsino. A short time after the issue of this paper, Dr. Looss received an appointment on the staff of the School of Medicine in Cairo. His next contribution to the Bilharzia question was published in 1905, as a paper [294] on the "Histoire Naturelle de la bilharzia," read before the "Premier Congrès égyptien de médicine":—

"Des expériences . . . faites par Cobbold, en Angleterre, par Lortet, en France, et par moi, en Egypte, eurent, sans exception, le même résultat négatif. J'avais simultanément examiné des centaines d'exemplaires des différentes espèces de mollusques égyptiens recueillis dans des endroits où la bilharziose m'avait été rapportée comme étant très commune. S'il y avait une cercaire de la bilharzia correspondant à celle des autres distomes, j'aurais dû la trouver, mais je ne découvris rien de semblable. J'ai finalement observé au microscope la conduite des embryons de la bilharzia en présence des mollusques sus-mentionnés ou de fragments de leurs corps: or tandis que les embryons d'autres distomes qui se développent en réalité dans des mollusques, sont attirés même par des débris encore vivants du corps de ceux-ci et s'efforcent d'y pénétrer, les embryons de la bilharzia, eux, ne prêtaient pas la moindre attention aux mollusque que je leur avais présentés."

"Toutes les observations mentionnées et d'autres encore, tendaient donc à établir que les mollusques ne peuvent pas jouer un rôle dans le développement de la bilharzia."

In a polemical article, "What is *Schistosomum mansoni?*" in the *Annals of Tropical Medicine and Parasitology* for 1908, Looss [295] again asserts:—

"All attempts made by former authors to discover an intermediary host in which this development is gone through have failed, and so have my own efforts. I have examined hundreds of specimens of all the molluscs common in the Nile Valley without finding any sporocyst which might have been brought into relation with the Bilharzia worms. I have placed quantities of

free-swimming miracidia in contact with the same molluscs without obtaining an infection. It is easy to infect molluscs with miracidia of species which actually develop in them. Bilharzia miracidia were never seen to take any notice of any mollusc in their neighbourhood, whereas others developing in a certain mollusc soon begin to swarm about it, and may, under the microscope, even be observed to enter into it. The same negative results were observed with larvæ of insects, with fishes, and with plants. I am thus forced to the conviction that man himself acts as an intermediary host."

"In man, the miracidium must develop into a sporocyst which either directly or indirectly generates the Bilharzia worms. The only organ of the body thus far known to harbour young, and sometimes very young worms, is the liver. I therefore conclude that the liver is the habitat of the sporocyst from which the worms later escape into the portal vein."

In 1909, in a controversial paper entitled "Bilharziosis of Women and Girls in Egypt in the Light of the Skin Infection Theory," Dr. Looss [296] replies dogmatically to some brief comments on the insufficiency of his hypothesis made by Drs. Elgood and Sandwith at the Annual Meeting of the British Medical Association:—

"Any theory about the mode of infection with bilharziosis, in order to be at all acceptable, must (*a*) account for the passages of the miracidium both from man to water and from water back to man: it must (*b*) duly consider both the habits of the host and the biological peculiarities of the parasite.

"The theory of miracidium infection by the skin is in accordance with all the facts thus far known (*a*) of the biology of the parasite, (*b*) of the distribution of the disease among the population (native and foreign, town and rural) of Egypt. It shows (*c*) how the chief sufferers—the children in town, the adult males in the country—live under conditions which, from the epidemiological point of view, are essentially the same, and give the miracidia (*d*) the opportunity of passing, within the short time of their life, from man to water and from water back to man."

In a popular lecture [297] on the "Life-history of the Bilharzia Worm," before the Cairo Scientific Society in 1910, Dr. Looss again declared:—

"What we know of this, the life-history of the parasite, is the following:—

"The worms which infect a fresh subject originate from the

eggs which have left the preceding host with the urine or the fæces. The only medium in which they find the condition for further development outside man is water." "Authors have tried to find a mollusc capable of rearing the germs of Bilharzia; I have personally experimented with all the species occurring in the Nile Valley, but in vain. I equally failed with larvæ of insects living in water, with various species of fish, with plants, etc. In such circumstances the only alternative left was to assume that the germs from the water immediately return into man, making their way to the liver, where they reproduce the *Bilharzia* worms."

During 1911-13 Japanese investigators had succeeded in infecting cattle, cats, and dogs with an allied disease, caused by *Schistosoma japonicum*, by immersing the animals in the flooded fields of infected areas, but had quite failed to obtain infection by similar immersion in water containing large numbers of live miracidia. Commenting upon this in 1914, Dr. Looss [300] says:—

"Merkwürdig ist dass die japanischen Autoren anscheinend zu keiner Klarheit über die Natur der Invasionsform kommen können." "Nach [Miyawa] zeigt die Invasionsform einen so beträchtlichen Unterschied" von dem Mirazidium, dass die Existenz eines Zwischenwirtes wahrscheinlich wird. Ist dies richtig, dann müsste sich *B. japonica* in ihrer Entwicklung wesentlich von *B. hæmatobia* unterscheiden, denn wie ein im Wasser lebender Zwischenwirt an der Verbeitung der letzteren in den Städten Ægyptens beteiligt sein soll, scheint zunächst schwer verständlich."

It is remarkable that the Japanese authors do not appear to be able to come to any clear concept as to the nature of the invading form. According to Miyawa, the invading form manifests such marked divergences from the miracidium, that the existence of an intermediate host seems probable. If this statement is correct,[1] *Bilharzia japonica* must differ essentially in its development from *B. hæmatobia*, for it seems *a priori* difficult to understand how an intermediate host that lives in water can participate in the spread of *B. hæmatobia* in the towns in Egypt.

Direct Infection Experiments.

The earliest attempts to obtain direct infection experimentally were made by Harley [216] in 1871. "Two young rabbits and two dogs were allowed to take, at intervals with their food, pellets of the mucus containing swarms of the eggs. Three of these animals were killed after an interval of two, three, and six months respectively, and carefully examined, but no trace of Bilharzia could be found."

Mantey is reported to have made some experiments in 1880, but his account [331] has not been accessible. The results are quoted by Looss as having been negative. Lortet and Vialleton

[1] This has been experimentally demonstrated by Miyairi and by Leiper and Atkinson.

attempted to infect guinea-pigs by passing the Bilharzia eggs into the stomach through a tube. They also injected the eggs into the saphenous vein of rabbits, and also attempted to infect a Macacus monkey by mixing large numbers of eggs in its food. These experiments were uniformly unsuccessful.

Having convinced himself that the Bilharzia did not require an intermediate host, Looss [288] attempted, in 1894, to infect monkeys by the mouth.

"Die Versuche wurden jetzt so angestellt, dass die ausgeschlüpften Embryonen mit filtriertem Wasser ausgewaschen und dann in reines, ebenfalls filtriertes Wasser übertragen und mit diesem an verschiedene Tiere zum Trinken gegeben wurden. Mit dem Menschen selbst zu experimentieren, ging leider nicht gut an; es würde, glaube ich, in Aegypten auch kein einwandsfreies Resultat ergeben haben, denn fast jeder zweite oder dritte Mann ist dort bereits von selbst infiziert. Ich benutzte deshalb Affen verschiedener Species, bei denen ja durch Cobbold ebenfalls das Vorhandensein einer Bilharz konstatiert wurde. Die Tiere bekamen durch 6 und 8 Wochen hindurch täglich ein, zwei, teilweise sogar drei Mal von dem stark embryonenhaltigen Wasser zu trinken, dasselbe wurde teils in gewöhnlicher Temperatur, teils auf 37° C. erwärmt gegeben einer Temperatur, bei der sich die Embryonen ausserordentlich lebhaft und agil zeigen—die spätere Untersuchung (nach 8 und 10 Wochen) mehrerer Affen ergab aber in allen Fällen ein durchaus negatives Resultat!"

The experiments were now carried out as follows: the freed embryos were washed in filtered water and then transferred to clean, likewise filtered, water, and this was given to various animals to drink. It was unfortunately impossible to experiment with the human being; and, indeed, in my opinion no incontrovertible results would have been obtainable in Egypt by such a method, considering that practically every second to third person is already infected. I therefore employ monkeys of various species, Cobbold having shown that a Bilharzia is found to occur in them. During from six to eight weeks the animals were given some of this highly embryo-infected water to drink once, twice, and in some cases even three times a day; the water was given either at the ordinary temperature or at 37° C., the latter temperature being one at which the embryos are found to be extremely lively and agile; subsequent examination of several of the monkeys (after from eight to ten weeks) yielded in every case absolutely negative results.

Skin Infection Theories.

The hypothesis that an unknown larval stage might enter the body through the skin had already been formulated in the *Practitioner* for 1888, by Allan [6], who wrote, "Nearly all the youths bathing in the Umzimdusi and Dorp Spruit are infected, while the girls, who do not bathe, remain free of the disease." In 1894 Brock [67] strongly supported this view: "My own inquiries have led me to the conclusion that

bathing is, in this neighbourhood, at any rate, the most fruitful source of the infection. I cannot, among several hundred instances, recall one exception to the rule that all who suffer from the parasite have been in the habit of bathing. Moreover, it is among boys, who are fondest of swimming, that the symptoms earliest make their appearance ; and I believe it would be hard, if not impossible, to find one boy much given to bathing in the streams, or their tributaries, who does not, before reaching manhood, become a subject of the disease. Only on this theory is explicable the fact noted by various observers in South Africa that the female sex is rarely attacked by *Bilharzia*.

"That the drinking of impure water is a common factor in the process of infection there is no lack of evidence ; indeed, it would be unreasonable to think otherwise if the bathing hypothesis be well grounded. But, other things being equal, the chances of infection occurring will be greater from the large quantity of water which must come in contact with the body in bathing, than from the comparatively small amount conveyed into the stomach by drinking ; so that, granting the larvæ to have the power of penetrating the body by some means, we should expect to meet with a much larger proportion of cases among bathers than among those who only drink the infective water.

"There is no evidence whatever that infection can take place by direct contagion. The question of how infection occurs will, however, only be satisfactorily solved when we have succeeded in tracing the development of the parasite through all its stages."

It was in an article (1894) criticizing this paper that Looss, while adopting the view taken therein that infection occurs through the skin, first maintained, on biological grounds, that the infective agent was the miracidium [288].

The biological facts upon which he based this deduction were explained by Looss [294] to the Egyptian Medical Congress in 1905, "il me sembla important de savoir si les embryons pouvaient, oui ou non, résister à l'influence des acides de l'estomac. J'ajoutai sur un porte-objet, au bord d'une goutte d'eau contenant quelques embryons vivants, un petit morceau de la muqueuse stomacale d'un singe récemment sacrifié et le comprimai légèrement pour en faire sortir quelque peu de suc. Dès que les embryons arrivaient à proximité de cette partie de la goutte, leurs mouvements cessaient presque instantanément. J'ai, plus tard, répété ces experiences avec un acide dont je connaissais la concentration. Une douzaine de larves, à peu près, ayant été recueillies dans un petit verre de

montre rempli d'eau pure dans laquelle ils nageaient aisément, une goutte d'une solution aqueuse d'acide hydrochlorique à un pour mille fut ajoutée avec précaution sur un côté du verre, mais sans mouvoir ce dernier. Aussitôt que les embryons, tout en nageant, entraient dans la zone acidulée, les mouvements de leur revêtement vibratile cessaient presque aussi vite que dans l'expérience avec le suc stomacal du singe. Les corps avaient encore quelques mouvements de contraction, mais au bout d'environ deux minutes les embryons étaient complètement morts. Peu à peu, huit embryons entrèrent de cette manière dans la zone acidulée et chez tous l'action de l'acide se montra absolument la même. L'expérience fut alors répétée, mais avec une solution d'acide à un pour deux mille. Même résultat que dans l'expérience précédente, avec cette simple différence qu'ici la mort définitive des embryons était un peu retardée.

" D'après ces expériences, il me semble impossible que les embryons libres, avalés avec la boisson, puissent franchir vivants l'estomac de l'homme. Vu ce résultat, il ne reste donc plus qu'une possibilité pour les embryons de pénétrer dans le corps humain ; de percer leur voie activement à travers la peau. Il y a quelques années que j'ai été conduit à cette conviction à la suite d'observations spéciales, et je peux dire que les observations occasionnelles que j'ai faites entre temps, ne m'ont que confirmé dans mon opinion."

" Malheureusement, je ne peux pas encore vous fournir la preuve incontestable par l'expérience. J'ai déjà entrepris quelques tentatives pour faire entrer les larves dans des animaux, mais ces expériences, peu nombreuses du reste, et exécutées sans système, n'ont pas donné des résultats décisifs."

The next attempt was made in German East Africa by Wolff, who failed to infect a cat by immersion for half an hour in water swarming with miracidia. His experiment is briefly recorded in *Archiv. f. Schiffs- und Tropenhygiene* for 1911 [521].

In 1912 F. E. Bour [51] wrote from Mauritius to the *Journal of Tropical Medicine and Hygiene* :—

" Four months ago an attempt was made to infect two monkeys (*Macacus cynomolgus*), one by keeping for one and a half hours in the hollow of his abdomen, closely shaved, some water containing numerous miracidia ; the other by dropping the same water into his mouth and on the mucosa of his lips and cheeks. Up to the present time no Bilharzia eggs have been detected in their urine."

In 1914 Conor [122] carried out an extensive series of experiments in Tunis to obtain direct infection. Miracidia were adminis-

tered to various species of monkey, to sheep, rabbits, guinea-pigs, and rats. Subcutaneous injection, applications to the shaven skin, attempts to infect by bathing and by the mouth, were without result in every case.

A number of experiments have also been made by Fülleborn, by certain of Dr. Looss's colleagues and by the author, but lacking success, these have not been put on record.

These repeated failures to obtain experimental verification of his theory are explained away by Looss [300] with the statement that man is the only known host of *Bilharzia hæmatobia*.

Dissentients.

It is right to add that a number of authorities, notably Blanchard and Manson, have consistently withheld their assent to the Looss hypothesis. In "Maladies parasitaires" Blanchard [37] wrote in December, 1895:—

"Il est hors de doute que l'embryon éclòt normalement dans l'eau et qu'il pénètre dans le corps d'un animal aquatique, pour y accomplir sa phase larvaire. Neanmoins on n'a pu jusqu'ici découvrir en quoi consistaient ses métamorphoses et chez quel hôte elles s'accomplissaient. Lortet et Vailleton . . . ont cherché l'embryon et la Cercaire dans les eaux des rivières et des mares d'Égypte au moyen de pêches au filet fin, sans pouvoir le rencontrer jamais. Ils ont dressé la liste des animaux qui vivent dans ces eaux et les ont examinés avec le plus grand soin, sans jamais y trouver aucun parasite qui soit imputable à l'une des phases de l'évolution de la Bilharzie. Le mode de développement et de propagation de ce Trématode reste donc encore entouré d'un profund mystère."

In 1905 Sir Patrick Manson [329], in his "Lane Lectures on Tropical Diseases" (p. 50), expressed the opinion that Bilharzia is "another illustration of the conveyance of a disease germ through water and probably by a fresh-water intermediary," while in the latest (1914) edition of his text-book on "Tropical Diseases" he writes: "Analogy suggests that the miracidium passes into the body of some fresh-water mollusc, crustacean, or larval arthropod, there to undergo the developmental changes in the redia and possibly the cercaria stages usually exhibited by the trematodes. Later, it may become encysted, and then, either free or still in the body of the intermediate host, gain access to man by penetrating the skin or through the stomach, and so pass to the veins of the portal system."

"Looss has expressed the opinion that, unlike other trematodes,

Schistosomum hæmatobium does not require the services of an intermediary host, and that the miracidium enters the human body directly by penetrating the skin." "I would remark that if *S. hæmatobium* does not require the services of an intermediary host its peculiar geographical limitations are difficult to explain."

In the following year Balfour [12] tentatively subscribed to the hypothesis that a minute crustacean was the essential carrier. He says: "In the first report the prevalence of the disease amongst the boys attending a primary school in Khartoum was mentioned. Many of these boys drank from the school well, and this water was submitted to examination. A tiny but very active entomostracean, probably belonging to the order *Ostracoda*, just visible to the naked eye, was seen.

"Six active embryos were placed in water along with three of the lively crustaceans and left over night. In the morning one dead embryo was found lying on the foot of the watch-glass, the other five had wholly disappeared, and the crustaceans remained alive and active. What had become of the missing five? Presumably they had entered or been taken up by the crustaceans."

Two years later (1908), in their "Review of Recent Advances in Tropical Medicine" [14], Balfour and Archibald add: "Time has not permitted further experiments with the species of Ostracode mentioned in the Second Report, but certainly the results obtained were suggestive."

Looss's Indications for Prophylaxis.

The practical requirements of his theory of direct infection are indicated in the following abstracts from his publications during 1908-14:—

(1908.) *Ann. Trop. Med.* [295]: "If this conclusion [infection by miracidia] is correct, it leads to the important consequence that the spread of the *S. hæmatobium* is not limited by the natural geographical distribution of a special intermediary host. It can spread wherever man carries it, so long as, and in so far as, the climatic and hygrographic conditions are favourable for its development." "The Egyptian peasants usually work their fields in companies; sometimes of two or three, sometimes of several dozens, standing with their feet, and working with their hands, in the water or the mud. They often also bathe in companies in canals with slowly flowing water, pools, etc. One of them who is infected with urinary bilharziosis, when urinating into the water, infects it with several hundreds, perhaps thousands, of eggs. In warm weather the miracidia hatch within a few minutes. They have at once the opportunity of finding a new shelter, either in the skin of the man

who voided the eggs or in the legs and hands of one of his comrades working close by him. . . . These possibilities of infection are repeated every time a man urinates into the water. They are perhaps repeated every day the season of the Nile flood lasts."

(1909.) *Brit. Med. Journ.* [296]: "When once free from their eggs' shells they [the miracidia] disperse in the water to all sides, and the chance of reaching a suitable shelter considerably decreases for the individual miracidium with time and distance. The more I think of this latter circumstance the more I become convinced that the chief foci of infection—that is, the places where strong and repeated infections are contracted—cannot be found in large bodies of waters, as rivers, canals, ponds, etc., but must be sought in small accumulations of water, in which the miracidia, once introduced, cannot become widely dispersed."

"To render an infection of the skin at all possible . . . the following conditions must be realized: An infected person must urinate (or defæcate) in a place where there is water, however small the quantity. The place must remain moist for some time, but not longer than thirty to forty hours. Within this period another person must bring some part of his skin for some time into actual contact with the moisture. If these conditions are fulfilled the miracidia have the *possibility* of getting from man to water and from water back to man; their life-cycle may be closed.

"The infective power of moist places *gradually decreases* and is *again nil* at the end of one or two days even if they remain moist. A recent contamination must take place in order to render them infective again for a short period."

"The moist places demanded for infection are to be found plentiful about town: in the streets there remain puddles for several days after each rain or for several hours after each watering; the courtyards of the houses also are often watered especially in the warm season. In many Arabic houses water-closets are an unknown institution or they are of the most primitive type. The calls of Nature are often obeyed in the streets, oftener in the courtyards, especially for urinating. There is thus sufficient occasion for the ground to become over and over again populated with live miracidia: their short life is of no consequence. There only remains the host to supply the miracidia and another to take them up again."

"Contaminated water loses its infective power again after having been protected from fresh contamination for one or two days."

(1910.) [297] "Taking the [miracidium] infection by the way of

the skin as an established fact, the measures to adopt for preventing it are clearly given. First of all, infected persons should never evacuate urine or fæces into water, for this is the only way in which the latter becomes populated with the dangerous germs. If any body of water can be shown to be safe from contamination, it must, as a matter of course, be left out of consideration as a source of infection. If water is likely to be, or is likely to have been, contaminated, it should not be used for bathing, washing, or working in before about two days have passed. But even here a certain reasonable discrimination should be made. For in a large body of (standing or flowing) water, e.g., the Nile, any germs are soon so much dispersed that the possibility of picking up one or another becomes very small and may be increased only by a very prolonged contact with the water. Dangerous in the first place are small bodies of standing water, both permanent and transitory, because in these the germs cannot disperse."

In the last edition of Mense's "Handbuch der Tropenkrankheiten" (1914) he claims :—

"Besteht die Haut infektion im Wirklichkeit (sie wird neuerdings von immer zahlreicheren Autoren als wahrscheinlichen bezeichnet) so ist zu beachten, dass eine Auto-Reinfektion auch bei Wannenbädern mögleich erscheint, wenn der Badende unter sonst gunstigen Bedingungen (absichtlich oder unabsichtlich) Eier in des Wasser entleer."

If cutaneous infection really occurs—latterly an ever increasing number of authors mention such an infection as probable—it is worthy of note that auto-reinfection appears to be possible also when taking baths indoors, supposing the bather, other conditions being favourable (either intentionally or unintentionally), voids the ova into the water.

Blanchard's views on prophylaxis are expressed in the following passage from his "Traité de Zoologie Médicale" [34], 1899 :—

"L'infestation se fait par les eaux de boisson, soit qu'on ingére l'hôte intermediaire lui-meme, et alors il s'agirait d'un mollusque de petites dimensions, soit plutôt qu'on avale la Cercaire nageant librement dans l'eau. . . ."

"On a supposé que le parasite pénétrait dans l'organisme à travers le peau et, par suite, on a interdit formallement les bains de rivière cette interdiction ne nous semble aucunement justifiée ; encore que nous ignorions les phases ultimes du développement, il y a disérieuses raisons d'admettre que l'Helminthe pénètre réellement par la voie que nous avons indiquée plus haut. C'est donc l'usage d'eau non filtrée ou non bouillie qu'il faut rigoureuse-

ment proscrire dans les pays contaminés ; l'usage des bains est indifférent."

In 1908 in a brief note, "Le renvoir d'un Collegien atteint de Bilharziose est-il legitime?" in the *Archives de Parasitologie*, he wrote:—

. . . "il serait donc très utile de savoir où le jeune homme habite ordinairement. . . . quelle eau il boit ordinairement : eau d'une source vive, d'un puits ou d'une citerne? Y-a-til dans cette eau des mollusques ou coquillages et peut où on obtenir des spécimens? De dernier point est de le plus haute importance.

La bilharziose n'est pas une maladie contagieuse. Il n'y a donc *aucune raison* pour refuser de garder au lycée un jeune homme qui en est atteint : il n'est aucunement dangereux pour ses camarades."

Manson's opinion on prevention, as given in his "Tropical Diseases" [328] in 1905, is as follows:—

"Since analogy justifies the belief that the embryo of Bilharzia on obtaining access to fresh water enters a fresh-water animal and by it obtains access to another human host, it is evident that if the embryo be kept from getting into the water or if drinking water be boiled or filtered, the spread of the disease from man to man would be effectually prevented. In the endemic districts children in particular should be carefully and repeatedly warned against drinking the water of ponds and canals. Provided reinfection be avoided by the exercise of prudence in the matter of drinking* water, there is no necessity for sending the patients with Bilharzia disease away from the country in which the parasite was acquired."[1]

In a popular lecture to the Rhodesia Scientific Association [330] in January, 1914, Sir Patrick Manson said, after briefly outlining the development of the liver-fluke : "In a similar way I believe the germ of Bilharzia disease, so common in this country, especially in young people and probably contracted in bathing in pools and rivers, is acquired and spread."

Incidence of Bilharziosis in Egypt.

The published statistics of the incidence of Bilharziosis in Egypt have been based mainly upon hospitals records. Reliable statistics are notoriously difficult to obtain in Egypt, owing to the ignorance

[1] In the fifth edition (1914) the above paragraph is slightly changed by the addition of the words "or bathing in" after "repeatedly warned against drinking" and by the deletion of the word "drinking" marked by an asterisk *.

and suspicion of the mass of the people and their deep-rooted objection to having their names or affairs set down in any paper or book that may form part of the Government archives. Such an ordinary chronic condition as Bilharziosis is frequently not thought worthy of mention in addition to any other disease the patient may wish treated or which may be the cause of death. There are certain beliefs, too, apparently considerably prevalent, which not infrequently account for concealment. Many cases, therefore, go entirely untreated and unrecognized.

Griesinger [201] found 32 per cent of infections in 363 autopsies made in Cairo up to 1856. Sonsino [453] obtained 46 per cent from 91 autopsies. In 1894 Kauffman [321] recorded from 500 autopsies that 369 males gave 40 per cent and 131 females gave 11½ per cent, i.e., a general average of 33·3 per cent with Bilharzia. Ferguson [168], in 1910, stated that his observations, based on considerably more than 1,000 post-mortem examinations at the Kasr Aini Hospital in Cairo, revealed the presence of this disease in no less than 40 per cent of Egyptian male subjects between 5 and 60 years of age. In 1905 Milton [346] showed that 930 patients were treated for Bilharzia as in- or out-patients at Kasr Aini Hospital during 1901.

In 1910 Professor Madden [321] published the figures from the annual reports of the Kasr Aini Hospital for the three years 1907-09, showing a total number of admissions for medical and surgical diseases 11,698, of which 1,270, i.e., about 10 per cent, had Bilharziosis. These cases represent only persons "suffering from pathological destruction of kidneys, ureters, bladder, urethra, and rectum, produced by severe and repeated bilharzial infections." Madden adds, "the mortality from Bilharziosis *per se* or its immediate complications was just over ten per cent; but this hardly gives one even an approximate idea of the real mortality, as many cases, when they do not appear to be improving, are taken out of hospital to die at home; all such cases are entered as unrelieved, but had they been left in hospital many of them would certainly soon have been included in the mortality tables." Professor Ferguson found that of a series of five hundred autopsies on male subjects eight per cent of all cases died of the effects of severe bilharzial infection.

In the course of the ankylostomiasis campaign during 1914-15 MacCallum obtained for the first time some exact and detailed information regarding the local incidence of vesical Bilharziosis in the provinces of Qaliubia and Sharqia. These results have not yet been published, but Dr. MacCallum very generously communicated

his statistics to assist the present inquiry. In Qaliubia, of 700 persons examined 44 per cent showed vesical Bilharziosis. In Sharqia, 66 per cent of the 1,089 patients attending the travelling hospital for ankylostomiasis at Minia el Qamh, and 70·9 per cent of 832 attending at Bilbeis showed Bilharzia eggs in the urine.

The Mollusca of Egypt.

The fresh-water molluscs of Egypt have been studied by a number of malacologists, and have been well described and illustrated, especially by Jickeli (1874) in "Fauna der Land und Süsswasser Mollusken Nord Ost Afrika's," by Bourguignat in various papers from 1863 to 1883, by Innes (1884) and Pallary (1909). The last writer, in his "Catalogue de la Faune Malacologique d'Egypte," summarizes very completely the work of previous collectors. Pallary's list (excluding land forms) is given in the adjoining table and in parallel columns are indicated: (*a*) Those species previously described in the works of Jickeli in 1879; (*b*) the species mentioned as having been examined as possible transmitters of Bilharzia in the writings of Lortet and Vialleton, Sonsino, and Looss, are marked respectively by the lettering "V." "S." "L."; and (*c*) the forms collected and examined in the course of the present inquiry. It will be seen that of the fifty forms catalogued by Pallary, Lortet and Vialleton mention only four, Sonsino specifies nine, and Looss eight. It will be noted that all the forms mentioned by Looss had been examined also by Lortet and by Sonsino. In the subjoining table is tabulated a complete list of the trematode larvæ hitherto found in Egyptian molluscs. A comparison of the finds of Sonsino and Looss shows that each investigator met with developmental forms overlooked by the others, and that the investigations of the present inquiry revealed a still larger number of new forms.

In the Natural History section of the magnificent "Description de l'Egypte" prepared by the authority of Napoleon during the French occupation in 1799-1801, eight of the commonest fresh-water molluscs are recorded and beautifully illustrated. In three of these we have found undoubted Bilharzia larvæ within half an hour's journey by tram and train from Cairo.

These are extraordinary facts in the light of Professor Looss's repeated contention during the past twenty years that he had examined hundreds of specimens of all the molluscs occurring in the Nile Valley without finding any developmental form which might have been brought into relation with the Bilharzia worm.

As every digenetic trematode hitherto investigated has been found to undergo its larval metamorphosis in a mollusc, the deductions made by Looss from investigations ranging over such a restricted part of the molluscan fauna of Egypt are entirely unwarranted. The conclusion of Lortet and Sonsino, that their inquiries had failed to reveal the essential invertebrate intermediary, was much more reasonable.

The Present Inquiry.

As the province of Qaliubia had been shown to be heavily infected with bilharziosis it was decided to make Cairo the head-quarters of the inquiry. It will be seen from fig. 1 that the province of Qaliub adjoins Cairo immediately to the north, lying between the eastern (Damietta) branch of the Nile on the west and the desert on the east, and is easily accessible by road and rail, being traversed by the highway and main line to Alexandria and their subsidiaries. At the request of Sir David Semple, Chief of the Public Health Department of Egypt, the vacant laboratories of the Professor of Parasitology in the School of Medicine in Cairo were kindly set apart for our use by the Director, and facilities for the keeping of experimental animals were provided at the Bacteriological Institute. The C.M.S. Hospital at Old Cairo constantly supplied fresh material for observation from their clinical cases. The programme of work laid down and consistently followed was :—

(*a*) To collect and specifically determine all the fresh-water molluscs in the selected endemic area, i.e., within half a day's journey from the laboratory in Cairo.

(*b*) To dissect large numbers of all species found for trematode larvæ.

(*c*) To differentiate among the larvæ found those showing the morphological characters peculiar to the bilharzia group.

(*d*) To ascertain which, if any, species of mollusc showed chemiotactic attraction for Bilharzia miracidia.

(*e*) To induce experimentally infection of animals brought from England with Bilharzia cercaria when found in the mollusc.

(*f*) To ascertain experimentally whether infection took place through the skin or by the mouth or in both ways.

(*g*) To ascertain experimentally the incubation period of the disease.

(*h*) To determine experimentally on infected animals the efficacy of medicines, reputed to be of value on clinical grounds, to destroy the bilharzia worms in the portal system.

EGYPTIAN SPECIES OF MOLLUSCA.

	Pallary, 1909	Jickeli, 1874	S., V., L., 1892-94	W.O. Mission, 1915
(1)	*Succinea cleopatræ*	+	..	+
(2)	*Physa acuta*	+	V.	+
(3)	*Physa subopaca*	–	..	+
(4)	*Limnæa Caillaudi*	–	..	+
(5)	(*var.*) *Raffrayi*	–	..	–
(6)	*Limnæa alexandrina* (= *natalensis*)	+	S. L.	+
(7)	*Limnæa truncatula*	+	..	?, +
(8)	*Limnæa pharaonum*	+	..	+
(9)	*Bullinus brocchii*	-	..	?
(10)	*Bullinus Dybowski*	–	..	+
(11)	(*var.*) *alexandrina* ..	–	S. L.	+
(12)	*Bullinus Innesi*		S.	+
(13)	*Bullinus* (*Isodora*) *truncatus* ..	–	..	+
(14)	*Bullinus* (*Isodora*) *contortus* ..	+	..	+
(15)	*Bullinus* (*Pyrgophysa*) *forskali* (= *Physa micropleura*) ..	+	S. L	+
(16)	*Planorbis* (*Menetus*) *boissyi* ..	+	..	+
(17)	*Planorbis* (*Menetus*) *alexandrinus* ..	+	..	-
(18)	*Planorbis* (*Menetus*) *poeteli* ..	+	..	–
(19)	*Planorbis* (*Menetus*) *Laurenti* ..		..	+
(20)	*Planorbis* (*Tropidiscus*) *philippi* ..	–	..	-
(21)	*Planorbis* (*Gyraulus*) *Ehrenbergi* ..	-	..	?
(22)	*Planorbis* (*Gyraulus*) *mareoticus* ..	+	S.	+
(23)	*Planorbis* (*Segmentina*) *letourneuxi* ..	–	..	–
(24)	*Planorbis* (*Segmentina*) *angusta* ..	+	..	+
(25)	*Ancylus clessini*	-	..	?
(26)	*Ancylus Isseli*	+	..	+
(27)	*Ampullaria ovata*	+	..	+
(28)	*Ampullaria kordofana*	–	..	–
(29)	*Ampullaria lucida*		..	–
(30)	*Ampullaria exigua*	–	..	–
(31)	*Ampullaria vitrea*		..	–
(32)	*Lanistes bolteni* (*carinatus*) ..		V. L.	-
(33)	*Vivipara unicolor*	+	S. V. L.	+
(34)	*Vivipara biangulata*		..	+
(35)	*Cleopatra bulimoides*	+	S. L.	+
(36)	*Cleopatra vexillata*	–	..	–
(37)	*Cleopatra verreauxi*	+	..	-
(38)	*Cleopatra cyclostomoides*		S. L.	+
(39)	*Bythinia goryi*	+	..	-
(40)	*Bythinia alexandrina*	-	..	–
(41)	*Bythinia letourneuxi*	–	..	–
(42)	*Bythinia pseudamnicola*		..	–
(43)	*Bythinia subbadiella*	-	..	
(44)	*Bythinia* (*Gabbia*) *semnaarica* ..	+	..	-
(45)	*Hydrobia erythræa*	+	..	
(46)	*Hydrobia stagnalis*	+	..	-
(47)	*Hydrobia* (?) *schweinfurthi* ..	+	..	
(48)	*Melania tuberculata*	+	S. V. L.	-
(49)	*Valvata nilotica*	+	..	+
(50)	*Neritina nilotica*	+	..	–

(*i*) To observe the conditions favourable and inimical to the life of the free-swimming cercaria and the effect thereon of acid solutions and other medicinal substances.

(*j*) To study the bionomics of the special molluscan intermediary, if obtained, for facts upon which prophylactic measures could be formulated.

An examination of the banks of the Nile to the south of and around Gezireh, an island opposite Cairo, showed that, excluding bivalves, there were very few living forms in the main stream. Similar results followed from an examination of the Ismailia Canal at Mataria, north of Cairo, and of the Giza Canal, on the western bank of the Nile.

A study of a map of the environs of Cairo showed that there was a large number of collections of water in the Zoological and Botanical Gardens at Giza. With the permission of the Director, Major Flower, these ponds were exhaustively examined and were found to contain nearly all the described species of molluscs, with one or two notable exceptions. With material from this source a type collection of molluscs was made so that specimens brought in later from infected areas could be rapidly compared and determined.

In the report of the ankylostomiasis campaign in Qaliubia it was noted that a small travelling hospital had been stationed at the village Qalama, thirteen miles north of Cairo and near the main road to Alexandria (fig. 1). Of ninety-five inhabitants of this village, forty-three had been found to harbour bilharzia. This small village seemed, therefore, at first sight, a suitable place in which to make an intensive study of the local molluscan fauna. It proved, however, more difficult of access than had been anticipated. There was, too, a very large pond or birket which could not be thoroughly examined. Finally, as will be noted from the map, the canal outskirting the village had passed through a number of other villages after it took off from the main stream. Infection acquired in Qalama might well have been due to infective larvæ carried on from a higher part of the canal, and not actually derived from molluscs at Qalama itself. It was consequently decided to seek a more suitable village, if possible (*a*) of easy access, (*b*) without a birket, and (*c*) on a small canal coming almost directly off one of the main supply canals.

El Marg and the Marg Canal.

In the meantime, the Arab who had been for many years attendant in the Department of Parasitology had volunteered to bring snails from a place where he had been in the habit of

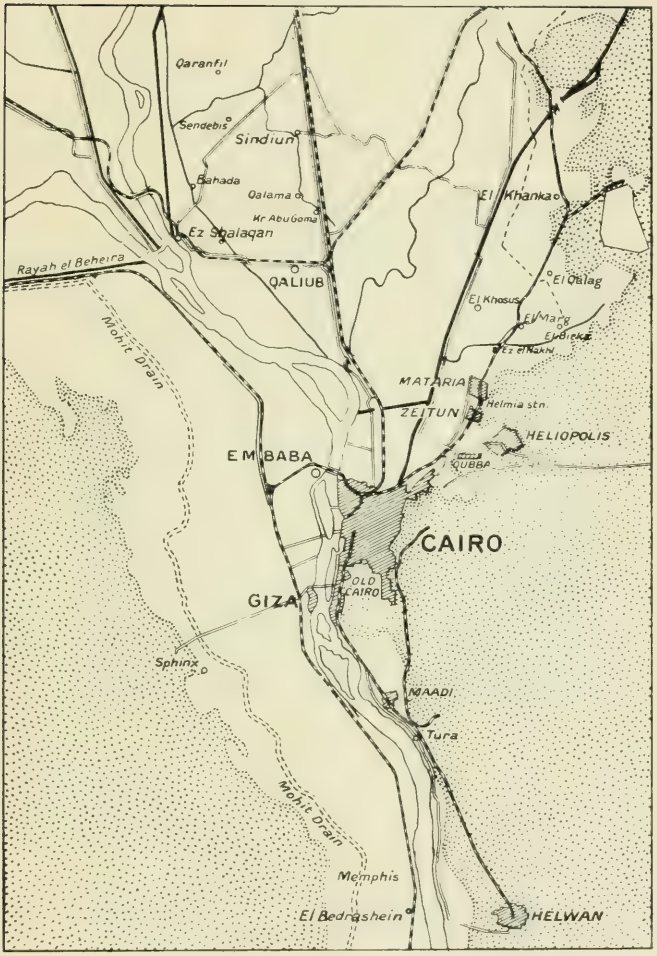


FIG. 1.—Cairo and the outlying villages.

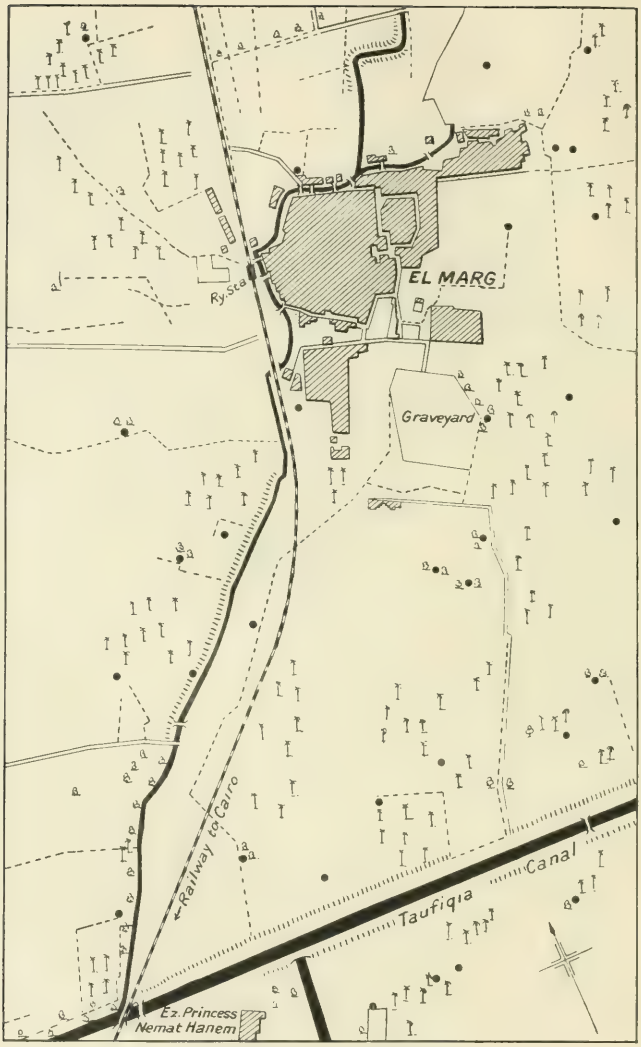


FIG. 2.—El Marg and the Marg Canal.

collecting them for the purposes of the class in zoology. His finds were both numerous and varied, and it was decided to prospect this collecting ground. It proved to be at his own village, El Marg, some nine miles north of Cairo, just beyond the popular and rapidly extending northern suburb of Cairo on the Zeitun-Mataria line. There was a good half-hourly train service, the village lay alongside the railway line and was an agricultural centre, being surrounded

Fig. 3.—Children from Marg village collecting water for domestic use.

by cotton fields and date palm groves. There was no birket, the only water supply to the surrounding fields was a small tertiary canal which traversed the village and derived its supply from the main Ismailia Canal a few miles distant, not far from its origin from the Nile. The village has a population of less than 5,000 and its inhabitants are mainly occupied in cultivating the surrounding land.

As will be seen from fig. 1, El Marg is situated in the centre of a cultivated plain, roughly triangular in shape, having as its base the Ismailia fresh water canal which runs north from Cairo, and

limited on the other two sides by desert. From the irrigation standpoint this triangle forms a small unit, the incoming water being drawn from the Ismailia Canal, mainly by the secondary Taufiqia Canal, which can be seen running eastward north of Mataria through Ez-el-Nakhl, where it is crossed by the railway from Cairo to El Khanka, and passing on through El Birka. The water from this canal and its subsidiaries, such as the El Marg Canal, after irrigating the land, drains into the large Bilbeis

FIG. 4.—Women washing garments in Marg canal.

drain, which runs northward from El Birka past El Qalag to Bilbeis, and is indicated on the map (fig. 1) by a broken line. The El Marg Canal takes off from the Taufiqia Canal near Ez-el-Nakhl station, and the flow is here controlled by an iron regulator. For about a mile the canal runs northwards parallel to the railway line, and on either bank there is a much frequented footpath. Just before the Marg railway station is reached, the canal passes under the line. At and above this point are the favourite spots from which the women and children carry water daily into the village for domestic use (fig. 3). After piercing

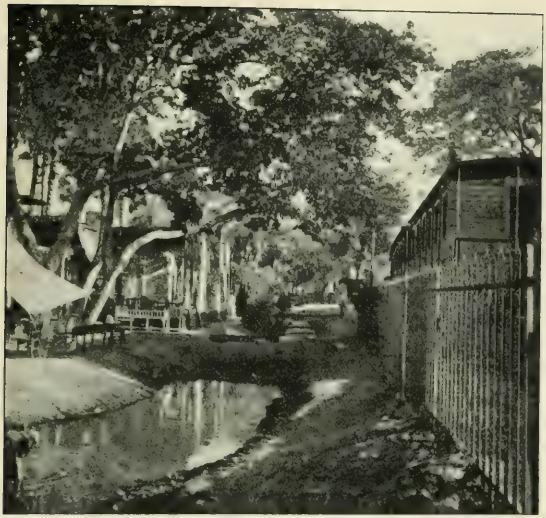


FIG. 5.—The canal skirting the village between the railway station and the cafés.

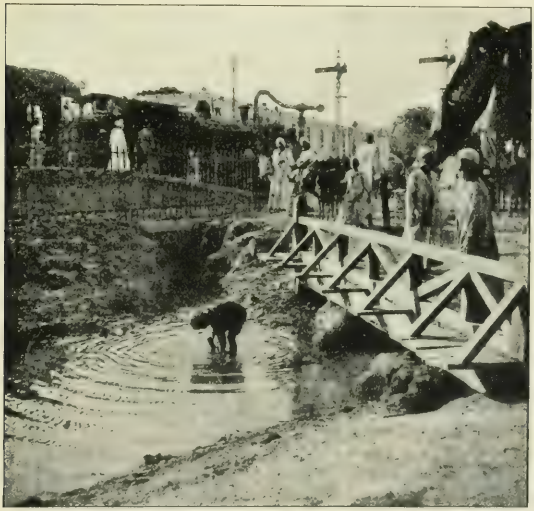


FIG. 6.—Marg bridge, crossing the canal from the station.

Fig. 7.—The canal forming a shallow pool in the village square.

Fig. 8.—Marg canal running northwards through the village.

FIG. 9.—The Marg Canal traversing the date palm grove.

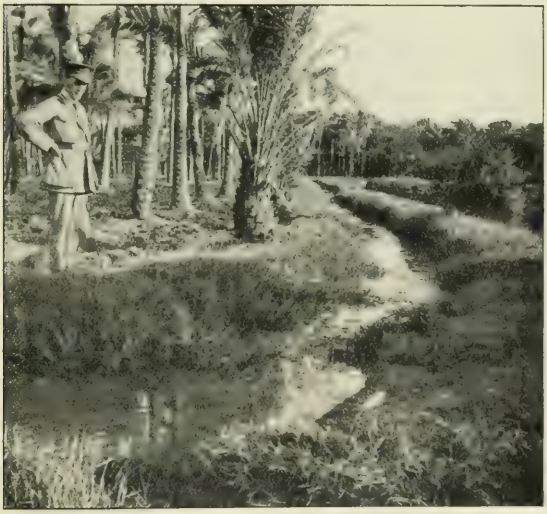


FIG. 10.—Marg Canal entering the cotton fields north of the village.

the railway embankment the canal continues alongside the line for about one hundred yards. This reach is much frequented by women for the purpose of washing clothes (fig. 4). The canal now skirts the houses, shops and cafés which front the railway station, and is open to constant contamination (fig. 5). A short wooden bridge crosses the canal at the station (fig. 6). Here the stream bends towards the centre of the village and forms a wide

Fig. 11.—A cul-de-sac in Marg canal dry during the "rotations."

shallow pool (fig. 7). Turning northwards again between two rows of houses, and with paths on either bank (fig. 8), it laces through a date-palm grove (fig. 9) and reaches the open country, where it rapidly diminishes (fig. 10) and ultimately divides into a number of small canals supplying the individual fields.

Incidence of Bilharziosis in Marg.

In the historical section reference was made to the frequent occurrence of bilharziosis among the French troops during the invasion of Egypt in 1799-1801, and it is of interest that the little village Marg was the site of one of the battles in 1799.

No statistics of the occurrence of bilharzia in this village were obtainable. A rapid examination of the urine of fifty-four boys in two of the village schools showed bilharzia eggs in forty-nine cases. The determinations were completed within an hour, and the positive results based upon single drops of freshly passed urine taken without either sedimentation or preliminary use of the centrifuge. Most of the boys were under 12 years of age, and had been born and reared

FIG. 12.– Marg Canal almost dry during a summer rotation.

in the village. It seemed reasonable to suppose that the bilharzia infection was derived from the Marg Canal, with its terminal branches and subsidiary agricultural drains around the village. It was decided, therefore, to make a complete census of all fresh-water molluscs in this the sole possible source of infection.

THE MOLLUSCA OF MARG.

A fortunate circumstance enabled us to make practically certain that no species living on or in the muddy bottom of the canal was overlooked. During the summer months the water coming from

the main canal is entirely controlled by the Government, and is supplied on a definite rota of six days' flow, succeeded by fifteen days' stoppage, in every three weeks. This rotation began early in April. During the fifteen days' stoppage the water entirely disappeared from the Marg Canal (fig. 11), save where inequalities left small puddles (fig. 12), and we were able to collect stranded shells throughout its length from the drying surface and to recover by sieving those forms that had succeeded in burrowing for some distance into the mud. The species found were : *Melania tuberculata, Vivipara unicolor, Cleopatra bulimoides, C. cyclostomoides, Bullinus dybowski, B. alexandrinus, B. contortus, B. innesi, Pyrgophysa forskali, Lanistes bolteni, Planorbis boissyi, P. mareoticus, Limnæa Caillaudi, Bythinia* (*Gabbia*) *sennaarica, Valvata nilotica* and some bivalves.

The commonest species were *Planorbis boissyi, Bullinus* and *Cleopatra, spp.*, the two first-mentioned being the most obvious forms on account of their habit of seeking the surface for air.

Technique.

The various molluscs, wherever and whenever collected, were separated into their different genera and, as far as possible, species, and examined for developmental stages : (*a*) by direct inspection, (*b*) by dissection. When an infected mollusc is kept in a glass vessel in clean water for a few days the cercarial forms are frequently discharged naturally by the mollusc, and may be easily seen swimming about in the water if the glass is held against the light and examined with a hand lens. By this method large numbers of the same species can be placed together in a vessel and tested without further technique. If, however, the larval development has not yet reached the stage for the discharge of the cercaria the results are apt to be misleading. Should a positive result be noted, the cercaria can be determined by microscopical examination and the infected mollusc isolated and kept alive for further observation. By dissection the earlier stages (sporocyst, redia, and immature cercaria) are obtained. The method has the disadvantage that the mollusc is necessarily killed in the course of the dissection and cannot be used for further work. The technique is simple though tedious when large numbers are involved. The shell, when hard and calcareous as in Cleopatra and Melania, is crushed with lion forceps, placed on a slide in a drop of water or weak formalin and the mass then torn apart with two dissecting needles. With the thin-shelled forms like Planorbis it suffices to fix the shell by piercing the central whorl with one

needle and to cut outwards with the second. In our experience the spear-shaped steel points issued for use on gramophone records when fixed in needle-holders are far more satisfactory than ordinary dissecting needles for this work. With them a large window can be quickly cut in the hardest shells and the molluscan body rapidly dragged out complete. The liver, which occupies the upper or central whorls of the gastropod shell, is the usual site of election for the developmental stages of trematodes. But a detailed examination of the mollusc is practically never necessary for the determination of an infection. With the first tear, as a rule, the characteristic bodies flood out in numbers into the surrounding plasma and water, while further search may be made under a low power by squashing the whole molluscan body slightly between two slides. In this way over 3,300 samples were minutely examined in the course of the inquiry. As shown in the appended list, seventeen species of Trematode larvæ were identified during these dissections. A number of new species were found also.

List of Trematode Larvæ identified in Egyptian Molluscs.

Molluscan host	Name of larva	Sonsino 1892	Looss 1896	Leiper 1915
Cleopatra	(1) *Gastrodiscus ægyptiacus*	–	+	+
	(2) *Cercaria distomatosa*	+	+	+
	(3) *Cercaria vivax*	+	+	+
	(4) *Cercaria exigua*		+	+
	(5) *Cercaria microcotyla*	+	–	+
	(6) *Cercaria capsularia*	+	+	–
	(7) *Cercaria cristata*	+	–	+
Limnæa	(8) *Cercaria obscura*	–	–	+
Bullinus	(9) *Cercaria pigmentata*	+	+	+
	(10) *Cercaria agilis*	+	–	+
	(11) *Cercaria fissicauda*	+	–	+
B. (Pyrgophysa)	(12) *Cercaria pigmentata*	+	+	+
Melania	(13) *Cercaria pleurolophocerca*	+	+	+
	(14) *Cercaria cellulosa*	–	+	+
	(15) *Cercaria monostomi verrucosum*	–	+	+
	(16) *Cercaria microcristata*	+	–	+
	(17) *Cercaria microcotyla*	+	–	+
Vivipara	(18) *Cercaria pusilla*	–	+	+
Lanistes	(19) *Cercaria sp.?*	–	?	+

Recognition of Bilharzia Cercaria.

The adult bilharzia worm resembles the other distome trematodes in possessing well-developed oral and ventral suckers, but differs in two remarkable ways: the sexes are separate, and there is an absence of a definite muscular pharyngeal bulb at the commencement of the œsophagus (fig. 21). As in all parasitic worms the infective

stage shows no sexual differentiation, consequently the absence of pharynx in the cercaria (fig. 19) is the one reliable character upon which a bilharzia cercaria can be distinguished from the cercariæ from other distomes, because the body of the cercaria without further metamorphosis grows to become the body of the adult worm. All cercaria have a motile tail at the posterior end. This appendage is a purely larval structure both morphologically and functionally. It serves the definite object of enabling the cercaria body to travel from the intermediate host to some favourable position where it can gain entrance to the final host. The tail is always shed before the actual arrival of the cercaria in the tissues of its host. Various cercaria exhibit different types of caudal appendage. In the four bilharzia cercaria that have come under the notice of the writer the tail was forked at its free end. This peculiarity, however, is shared with some other widely different forms among the distomes. It will be shown in the latter part of this report that four distinct sub-groups of bifid-tailed cercaria occur in the mollusca of Egypt.

Attraction of Mollusca for Bilharzia Miracidia.

Looss states that none of the Egyptian mollusca exhibited the slightest attraction for the freshly hatched miracidia of bilharzia. Most of the species submitted to experiment by us were entirely ignored by the miracidia. A definite attraction, however, was exhibited by the following: *Planorbis boissyi*, *Bullinus sp.* (?), *Pyrgophysa forskali*, and *Limnæa truncatula*. The attraction was stronger in young specimens. This plurality of susceptible forms appeared to indicate the possibility of a plurality of intermediate hosts or the susceptibility of the *Bilharzia hæmatobia* miracidia to the intermediate hosts of other species of bilharzia, or merely, as seemed probable in *Limnæa truncatula*, to a peculiar adhesiveness of the mucus covering the exposed portion of the mollusc body.

It may be noted here that not a single specimen of *Limnæa truncatula* could be found in or around Marg.

Marg Molluscs found infected with Bilharzia.

Large numbers of snails collected from the Marg Canal throughout its course, but more especially within the village, were infected with larval forms showing the morphological peculiarities of the bilharzia group. The infected shells were readily obtained at spots daily frequented, such as the praying ground at the embankment

crossing (fig. 11), in front of the cafés (fig. 5), and at a bend in the canal specially used for washing, which is illustrated in fig. 10.

The same species of mollusc was quite common at some distance from the village in the agricultural drains away from foot-paths, but was not infected. Nearer the village, however, and especially where crossed by public paths, these drains contained infected snails (fig. 13). So far the molluscan intermediary has

Fig. 13. A typical agricultural drain on the outskirts of Marg, containing infected snails.

not been found in birkets, but it occurs not uncommonly in large marshes such as that, lying to the south-west of Ismailia, which is illustrated in fig. 14.

Once a bifid-tailed cercaria has been placed in the bilharzia group on account of the absence of pharyngeal bulb, further determination of its systematic position can only be effectively established in the first instance by experimental infection of a susceptible host and the subsequent examination of the adult worm resulting therefrom.

Attempts to Infect Animals Experimentally.

Three cercariæ of bilharzial type were found in four different species of molluscs. The first form met with differed considerably from the other two, more especially in possessing a pair of black pigment spots just anterior to the ventral sucker, and a delicate but well-defined cuticular expansion on each side of the two prongs of the bifid tail. This form was found exactly five weeks after the

Fig. 14.—Marsh, near Kosti village, south-west of Ismailia.

commencement of our search, and occurred in *Planorbis marcoticus* collected from the ponds in the Zoological Gardens at Giza. Apparently identical cercariæ were found later at Giza and Suez in *Melania tuberculata*, and at Ismailia in *Planorbis boissyi*. The second bilharzia cercaria occurred in large numbers in *Planorbis boissyi* at El Marg, and was found later in similar canals between Inshas and Bilbeis in the Sharqia province. A very few cercariæ, apparently identical with this, were recovered on one occasion only from a *Melania tuberculata* in the Zoological Gardens. A third

cercaria, which on morphological grounds was only provisionally distinguished from that occurring in *Planorbis boissyi*, was also found at Marg in a certain number of specimens of *Bullinus* (fig. 15, A).

At Marg the *Planorbis boissyi* (fig. 15, B) were so commonly infected with non-eyed *Bilharzia cercaria* that half-an-hour's collecting sufficed to ensure a large supply of active larvæ. An extended series of experiments was instituted to determine the specific character of these forms.

Three species of *Bilharzia* worms are supposed to occur in Egypt: In man the *Schistosoma hæmatobium* (both varieties), in cattle the *S. bovis*, and in ducks the *Bilharziella polonica*. As bifid-tailed cercariæ with eye spots have been found, though not identified, in snails both in Central Europe and in North America, and as this cercaria departed somewhat from the two other forms, it seemed a reasonable conjecture that this cercaria was the larval

A B

FIG. 15.—A, *Bullinus cortortus*; B, *Planorbis boissyi*.

stage of an avian *Bilharzia*, and that the two remaining and similar cercariæ probably attained their maturity in the two known mammalian hosts. Attempts were made to verify this conjecture experimentally. In order to exclude possible fallacies it was essential to infect ducklings immediately after hatching and before they had come in contact with other than filtered water. These were obtainable in Cairo.

One was struck by the apparent entire absence of rats in the banks of the canals and in the fields. The authorities at the Giza Zoological Gardens stated that the common water-vole does not occur in Egypt. A professional rat-catcher was commissioned to obtain rats and field-mice from around Marg. His search proved quite fruitless. It was possible that this extraordinary absence of rodents might be due to a susceptibility to Bilharzia disease.

The animals frequenting the canal at Marg were man, cattle,

sheep, dogs (possibly rats), geese, ducks, chickens, and crows. Man and cattle were the most obvious sources of contamination of the canal water, and as each was a known host of species of *Bilharzia*, the probabilities were that cercaria normally developed in them.

Attempts to infect a calf and a lamb by allowing water heavily charged with the living cercaria to remain in the hollows of the groin for periods of ten to thirty minutes on several days gave entirely negative results at the post-mortems some weeks later. It was noted, however, in the case of the lamb, that the skin, where repeatedly exposed to infection, became markedly red.

A series of experiments was then made on mice, rats, geese, ducks, chickens, crows, and wagtails. The experiments on the birds proved entirely negative.

A positive result became apparent by June 13 in a young white rat, which had been infected on May 4. A black mouse which had been infected on May 2 died on June 24 with a number of Bilharzia worms in the liver and mesenteric veins.

In these early successful infections the mice and rats died from the occlusion of the portal system before the Bilharzia worms had reached sexual maturity. A comparative study of mature specimens of *Schistosoma bovis* and *S. hæmatobium* showed that these two species are so closely allied that, when experimentally reared in an abnormal host, a differential diagnosis could only be made with certainty upon the characters of the fully grown worms, and especially upon those of the egg shell. Further experiments were made. In those animals which survived for seven to eight weeks, female worms were found containing the characteristic eggs (fig. 20). This placed the diagnosis beyond question.

The animals used were variegated mice and white rats brought from London and fed solely on oats and filtered water. They had been kept in the laboratory under conditions entirely precluding the possibility of unobserved infection.

Animals found Susceptible to Infection.

In addition to tame white rats and variegated mice, the Egyptian desert rat, obtained from the neighbourhood of the Pyramids, was found to be susceptible to experimental infection, while guinea-pigs were peculiarly so. Mangaby monkeys died of acute bilharziosis within two months of infection. Experiments were not made on dogs owing to the quarantine difficulties that would have arisen on our return to England. At the conclusion of its field work the Mission

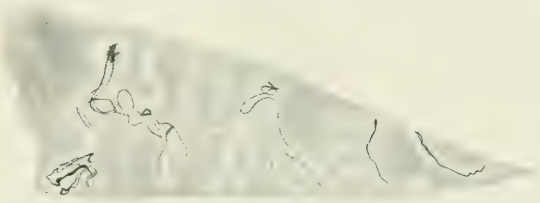


FIG. 16.

Edge of liver of mouse experimentally infected with bilharzia.

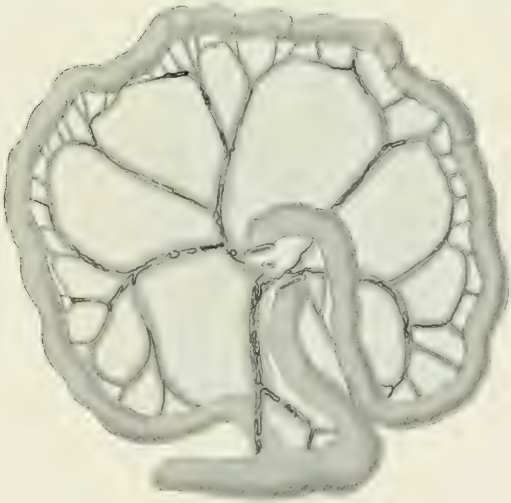


FIG. 17.

Mesentery of mouse experimentally infected with bilharzia.

To illustrate "Report on the Results of the Bilharzia Mission in Egypt, 1915," by Temporary Lieutenant-Colonel ROBERT T. LEIPER, D.Sc., M.B., R.A.M.C.

FIG. 18.

Section of liver of white rat experimentally infected with bilharzia showing veins blocked by worms.

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brought back from Egypt four mice, twenty-six white rats, sixteen desert rats, two guinea-pigs, and four mangaby monkeys, which had been submitted to infection shortly before departure. When examined shortly after their arrival in England all these animals had enormous numbers of bilharzia worms in the portal system.

Effect of Weak Acid and Alkali on Living Miracidia and Cercaria.

The theory that Bilharzia infection takes place through the skin was put forward by various South African writers from Harley to Brock, because in their clinical experience infection was found to follow repeatedly upon bathing in heavily contaminated waters. Looss, on the other hand, adopted the view because weak acids were found to kill the miracidium and consequently this, his hypothetical infective agent, could not survive in the stomach when taken in drinking water. The favourable action of weak alkalis and the destructive effect of dilute acids on all cilia is one of the recognized exercises in practical physiology. That alkalis and acids should have a similar effect on the ciliated body of the miracidium may be regarded therefore as a foregone conclusion. It does not necessarily follow, however, that the same result would obtain in the case of cercaria which are covered with a cuticular skin. Experiments were undertaken therefore to ascertain exact information on this point. Looss and others have shown that miracidia are killed immediately by a dilution of 1 in 2,000 of hydrochloric acid. Cercariæ withstand hydrochloric acid 1 in 880 for five minutes; 1 in 500 kills Bilharzia cercaria immediately. The acidity of the stomach would therefore inhibit these cercaria. This does not exclude the possibility, however, that infection may take place through the mouth. The ankylostomes and strongyloides infect through the mouth as well as through the skin. Fülleborn has recently shown that when the strongyloides are taken in by the mouth they do not pass on directly in the lumen of the gut into the small intestine, but penetrate the wall and, gaining the blood-stream, follow the circuitous route through the lungs up the trachea and then pass for a second time down the œsophagus through the stomach to the small intestine.

Mode of Entry of Bilharzia into the Body.

That something similar to this might occur in Bilharzia was suggested by a simple observation on the habits of the Bilharzia cercaria. These cercaria progress by swimming mainly through the

activity of their tails or by creeping by means of their well-developed suckers. When actively swimming cercaria are poured from one dish into another it is very noticeable that they immediately fix themselves firmly by means of their ventral suckers to the surface of the vessel into which they are carried until the motion of the water has ceased, when swimming is again resumed. It seemed, therefore, quite possible that if heavily infected water was drunk, the cercaria, coming into momentary contact, might similarly adhere to the mucous membrane of the mouth, tongue and œsophagus and at once proceed to penetrate into the tissues. That this actually can take place is apparently borne out by the following experiment. Four sooty monkeys which had been taken out with the Mission in January and kept in separate cages in the laboratory until the experiment was completed, were subjected to infection with Bilharzia cercaria. In three cases the infected water was poured into the bottom of the cages and the monkeys were consequently exposed to infection through the skin of the hands, feet, buttocks and tail. The fourth monkey was fed for a day or two on dry food only, and then allowed to drink from a cup containing food and water swarming with Bilharzia cercaria. Some effect, probably a prickly sensation, was produced almost immediately, for the monkey began to pull down the lower lip, to rub the mucous membrane of the mouth, and in other ways to indicate that the drink had not been pleasurable. After a second experience on the following day the monkey refused to accept water out of a cup although thirsty. The four monkeys eventually died and all showed a heavy infection with Bilharzia. The monkey infected from drinking water showed earlier and much more intense symptoms than the others. This experiment showed that in Bilharzia infection may be both oral and cutaneous. As in ankylostomiasis there is little doubt, however, that the infection enters through the skin in the bulk of cases.

As Brock [67] has pointed out, the chances of infection are much greater in bathing than in drinking, because under the former circumstances a much larger quantity of water comes into contact with the body.

Incubation Period of the Disease.

The incubation period of Bilharzia has been variously estimated by different writers. Sonsino gives from two to three years. Sandwith considers that it ranges from three to six months.

Hatch [222] in 1887 wrote : " The time between the contraction

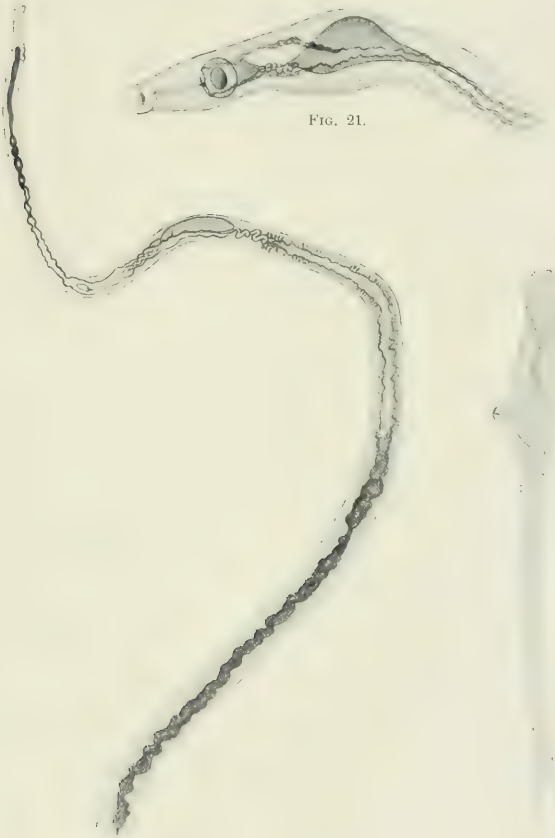

FIG. 21.

FIG. 20.

FIG. 19.

Fig. 19.—Cercaria of Bilharzia used in experiment. × 200.
Fig. 20.—Adult female taken from portal vein of mouse, experimentally infected mouse. × 18 (circa).
Fig. 21.—Anterior portion of adult male taken from the same source. × 18 (circa).

To illustrate "Report on the Results of the Bilharzia Mission in Egypt, 1915," by Temporary Lieutenant-Colonel ROBERT T. LEIPER, D.Sc., M.B., R.A.M.C.

of the disease and the passage of blood and other symptoms may be very short; one patient, who stayed at an hotel at Suez for fourteen days, suffering from them a month after his return to Bombay." More extensive observations were made during the South African War. Beveridge [24] gives the following valuable table in the JOURNAL OF THE ROYAL ARMY MEDICAL CORPS for 1907:—

Case	Age	Service year	Date of arrival in South Africa	Date of reporting sick with Bilharzia ova in the urine	Interval between arrival and reporting sick	Remarks
1	20	1	9.11.02	1.2.03	12 weeks	
2	20	1	9.11.02	17.2.03	14 ,,	
3	20	1	9.11.02	6.2.03	4 months	
4	21	1	9.11.02	19.3.03	4 ,,	
5	20	1	9.11.02	20.3.03	4 ,,	
6	23	3	9.11.02	6.7.03	7 ,,	Gives a history of 4 months' duration.
7	16	1	4.12.02	19.6.03	6½ ,,	Gives a history of 3days' duration.

In the same year Dr. Stock [475] wrote: "The time which elapses between its entrance and the onset of symptoms seems to be settled from the observations of Dr. Abercrombie. During the time that the regiment was at Pretoria several young drafts were sent out direct from England and all in due course bathed in the spruit, and as a result several cases occurred in young soldiers whose residence in the country had not been more than two months. The shortest period observed was one month, and the longest two months."

A similar incubation period occurred in the monkeys infected by us experimentally. Monkey No. 1, infected by immersion on June 15, 17, 24, and July 11, passed bilharzia eggs on August 4 and died on August 18. Monkey No. 2, immersed on June 17, 20, 23, and July 11, passed blood and mucus containing bilharzia eggs on July 30 and died August 9. Monkey No. 3, immersed June 17, 20, 23, and July 11, showed bilharzia ova in the wall of the large intestine after death on August 2. Monkey No. 4 drank infected water June 24, and on other occasions, including July 11, passed schistosome eggs and blood on August 4 and died of acute bilharziosis on August 8.

Occurrence of Bilharziosis in Large Towns.

There is one argument brought forward in his numerous papers by Professor Looss which seems at first sight to lend real support to his view that the mollusc is not required for the bilharzia

worm; that is, the accepted occurrence of bilharzia infection among children born and bred in large towns such as Cairo, where there is a filtered water supply.

Looss states that 33 per cent. of the boys attending a school in Cairo showed bilharzia eggs in the urine, and in 1908 Mrs. Elgood [158] showed that out of forty girls aged 12 to 16 in a middle-class school in Cairo, 11 (i.e., 27·5 per cent) were infected. These girls with one exception had lived all their lives in Cairo. None had ever bathed in the Nile or a canal, nor had any ever run about

Fig. 22.—Courtyard of Department of Physical Science, Cairo, showing supply of unfiltered water pumped direct from the Nile.

bare-legged in fields or country roads. The general water supply in Cairo is the same for natives as for Europeans, is of high quality and is supplied from the filters of the Cairo Water Company. These facts have been repeatedly used by the supporters of the Looss hypothesis, and certainly if correct could scarcely be otherwise explained.

After some inquiry the following facts came to light, which seemed to afford a simple and adequate reply to this objection. In

addition to the series of pipes supplying Cairo with filtered water, it appears that there is a second system carrying to the numerous gardens of Cairo unfiltered water drawn direct from the Nile in the neighbourhood of the Kasr Nil bridge, a spot where, in recent years, numbers of European troops have, while bathing, become infected shortly after their arrival in Egypt. It is well known that the children, even of the better-class Egyptians, are allowed to run about in the privacy of their own courtyards in a state of semi-nudity during the summer months, and are thus continually exposed to the risk of infection from the hose used in the garden or stable. The lower classes probably derive their infection from the same source, although under different circumstances. To them water is a dear commodity in Cairo. There is no free supply. In the poorer quarters one frequently sees water being hawked about in large skins, and there is the standing inducement to the middleman to increase his margin of profit by arranging to draw his stock, possibly surreptitiously through a friendly gardener, from the unfiltered supply for which the water companies make a lower charge. At Ismailia there is a dual supply of filtered and unfiltered water into the houses, the latter being laid on to the bathroom and kitchen and supplied at a lower rate. A standard pipe from the unfiltered supply in Cairo is shown in fig. 22 from a photograph taken in the courtyard of one of the Government offices.

Conclusions Contrasted.

Conclusions based on the Looss Hypothesis.

(1) All transient collections of water, such as those resulting from occasional showers of rain, road waterings and domestic waste, are dangerous if freshly contaminated.

(2) Large bodies of water, such as the Nile, canals, marshes and birkets, are little liable to be infective.

(3) All water in a given area would automatically become safe in thirty hours if the native infected population were removed.

(4) Infected troops would be liable to reinfect themselves, to spread the disease among other troops, and to convey the disease to any part of the world.

(5) Infection only takes place through the skin.

(6) Infection in towns is due to contact with recently contaminated moist earth or water.

(7) Eradication depends upon education and complete sanitary control throughout the country. The sustained co-operation of the affected individual is essential.

CONCLUSIONS BASED ON THE RESULTS OF THE PRESENT INQUIRY.

(1) Transient collections of water are quite safe after recent contamination.

(2) All permanent collections of water, such as the Nile, canals, marshes and birkets, are potentially dangerous, depending upon the presence of the essential intermediary host.

(3) The removal of infected persons from a given area would have no effect, at least for some months, in reducing the liability to infection, as the intermediate hosts discharge infective agents for a prolonged period.

(4) Infected troops cannot reinfect themselves or spread the disease directly to others. They could only convey the disease to those parts of the world where a local mollusc could efficiently act as carrier.

(5) Infection actually takes place both by the mouth and through the skin. Recently contaminated moist earth or water is not infective.

(6) Infection in towns is acquired from unfiltered water which is still supplied, even in Cairo, in addition to filtered water, and is delivered by a separate system of pipes.

(7) Eradication can be effected without the co-operation of infected individuals by destroying the molluscan intermediaries.

(*To be continued.*)

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(*To be continued.*)

INJURIES OF THE SUPERIOR LONGITUDINAL SINUS.

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Symptoms of Injury of the Longitudinal Sinus.

Experiences in the present War have made us acquainted with many conditions which are rarely seen in civil practice and have especially presented to us groups of uncommon neurological symptoms or clinical pictures; these are also often less complicated and more sharply defined than those due to the ordinary pathological lesions, with which we were previously familiar.

The vascular lesions of the brain met with in civil life, for instance, are most commonly due to arterial disease, to thrombosis, hæmorrhage, or embolism, while primary affections of the cerebral veins are uncommon; on the other hand, in gunshot injuries of the head, especially when tangential or superficial, disturbance of the cerebral venous circulation by depression of fragments of the skull is frequent, owing to the superficial course of the cerebral veins and the fact that their thinner walls and the lower pressure of the blood that flows through them makes them more liable to be blocked by pressure than the arteries.

The most striking clinical effects are, however, produced when the cranial sinuses into which these cerebral veins flow are affected, and in our experience much the most common of these to be involved is the superior longitudinal sinus. This receives on either side the veins which drain the mesial aspect, as well as those which carry blood from the superior half of the lateral surface of each hemisphere; while the veins from the lower part of each lateral surface pass through the sylvian system which opens directly or through the sinus spheno-parietalis into the cavernous sinus. There is often, however, a fairly free anastomosis between these two sets of lateral cerebral veins, and consequently complete occlusion of the one set may not necessarily produce permanent blocking

of the venous outflow from the area naturally drained by it. A sudden blockage may, however, lead to circulatory disturbance sufficient to abolish, temporarily at least, the functions of a part of the area normally drained by the venous system affected.

The manner in which the superior cerebral veins open into the longitudinal sinus is important in relation to the symptoms produced by lesions in its neighbourhood. This arrangement has

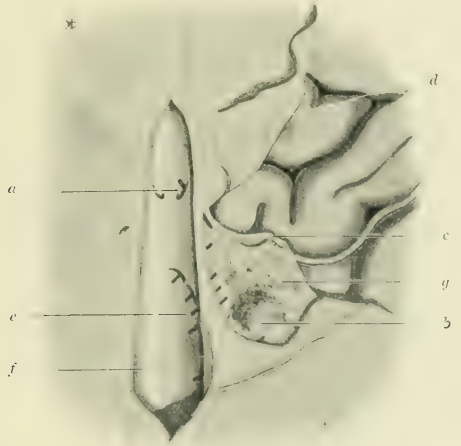

FIG. 1.—Dissection of the parietal lacuna on the right side, showing the opening of the post-central vein into the lacuna, and of the lacuna into the longitudinal sinus by six apertures. *a*, superior longitudinal sinus laid open by cutting away its roof; *b*, a lateral lacuna, laid open in a similar manner, showing a Pacchionian tuft projecting into its floor, and at *c* the valve-like opening of a large cortical vein; *d*, dura mater turned back to show the cerebral convolutions; *e*, glass rods showing the channels by which blood reaches the sinus from the lacuna; *f*, rod passed through a forwardly directed opening; *g*, a large Pacchionian tuft.

been fully described by one of us (P. S.). Although there is no strict constancy the superior lateral veins usually unite into four principal trunks, a frontal, a pre-central, a post-central, and an occipital; of these the post-central is usually the largest, and, as it drains the central gyri, the most important. As a rule these veins do not open directly into the sinus, but into thin-walled lacunæ

that project from it over the lateral as well as over the mesial surface of the hemisphere. A small frontal lacuna receives the frontal vein, a large parietal lacuna the pre- and post-central veins, and an occipital lacuna the occipital vein. Sometimes these lacunæ are more or less continuous. The larger veins either open directly into the floor of the lacuna, from which the blood finds its way into the sinus by several small openings, or they may pass beneath the lacuna and open directly into the longitudinal sinus generally supposed, and may spread at least two and a half centi- (fig. 1). The parietal lacunæ are often more extensive than is metres on to the convexity of the hemisphere, consequently any depressed bone or direct injury in their neighbourhood within this distance of the middle sagittal line may block the venous circulation of the upper parts of the central gyri, and owing to the thinness of walls this is more easily produced by pressure on the lacunæ than on the rigid sinus.

Different groups of symptoms are produced by lesions of the different lacunæ, but we intend to limit our description to those due to disturbance of the circulation through the parietal lacunæ, when they are predominantly those of disturbance of motion and sensation.

We have up to the present seen over seventy cases in which the longitudinal sinus was injured, or the circulation in its venous tributaries disturbed in its immediate neighbourhood, i.e., in the lateral lacunæ, or where the veins enter it. In many of these cases there was no direct damage to the brain, either by the projectile, or by depressed fragments of bone, but in others the disturbance of the venous circulation was associated with gross cerebral lesions.

The symptoms in these cases have naturally varied very much, according to the severity of the injury and the region in which the sinus was damaged, but the chief features of the condition can be best conveyed by describing a typical case; other types and other symptoms which occur will be considered later.

Case 1.—Private J. H. was wounded by a bullet on December 12, 1914. He became unconscious at once and was unable, on admission to the Base Hospital two days later, to give any accurate information about himself. He was dull and apathetic but answered questions quite readily.

There were two separate penetrating wounds four centimetres apart and equidistant from the midline, and about seventeen centimetres behind the nasion, that is, slightly behind the midpoint. The entrance was on the left; the exit on the right was slightly larger and slightly anterior to it; the skull between them was comminuted and some

disintegrated brain escaped through the exit. His speech and the functions of all his cranial nerves were unaffected, but the retinal veins were swollen and the inner margins of the optic discs were blurred and indistinct. His arms lay adducted to his side, flexed and pronated at the elbows, and were very rigid at the shoulders and elbows, but only slightly so at the wrist and in the fingers. He was unable to perform any voluntary movement with the right, but could flex and extend the left fingers feebly. The abdominal muscles were rigidly contracted and his respiration was mainly thoracic. Both lower limbs were very rigid and fully extended at hips, knees and ankles, and rotated inward and adducted at the hips so that the patellæ came in contact with one another; owing to their position and their extreme rigidity they resembled strongly the lower limbs of a severe case of Little's disease. This extensor rigidity was not constant, as occasionally the limbs were found rigid in flexion, but as a rule it was so great that the limbs could not be passively flexed or separated from one another by any reasonable force. The knee- and ankle-jerks were much exaggerated, and the hamstring-jerks were present and brisk; the flexor-jerks in the arms were also exaggerated but the triceps-jerks were feeble; both plantar responses were extensor and the abdominal reflexes were absent. When admitted his mental state was too dull to permit a proper examination of sensation.

Three days later he had a prolonged left-sided Jacksonian fit which commenced in the face. Ten days after receiving the wound he showed definite signs of improvement; his lower limbs were still completely paralysed and extremely rigid, fully extended, adducted and rotated inwards, but he was now able to move his fingers freely, and perform feeble movements at the elbows; the shoulders were, however, still rigid and their movements completely paralysed.

Twelve days later all movements of the upper limbs were possible, but the proximal muscles, especially of the right, were very feeble and all efforts he made were very ataxic; both arms were still rigid at the shoulders, and the right at the elbow, and constantly lay closely adducted to the sides and flexed. The legs, too, remained extremely rigid, extended, adducted and rotated inwards, but he occasionally had strong flexor spasms in them, especially when turned on his side. No definite voluntary movement of either was possible and any effort only resulted in a general contraction of all their muscles, and a slow, vigorous extension if any segment of the limbs were flexed. Stimulation of either sole produced a vigorous withdrawal movement of the limb without any contralateral effect. All the tendon-jerks were greatly exaggerated, the plantar responses were of the Babinski type and the abdominal reflexes were abolished.

The examination of sensation revealed, especially on the right side, the disturbances found in pure cortical lesions when the stage of shock or diaschisis has passed; the lightest touches could be appreciated

normally but a certain percentage of purely tactile contacts failed to evoke a response, and this failure bore no definite relation to the intensity of the stimulus. Localization of touch stimuli was, however, not seriously disturbed. There was no diminution to painful stimuli and no definite subjective difference in pin-pricks between normal and possibly affected parts. The appreciation of position and of passive movement was almost completely lost in both lower limbs and in the right arm, but was little affected in the left arm, and corresponding thereto the discrimination

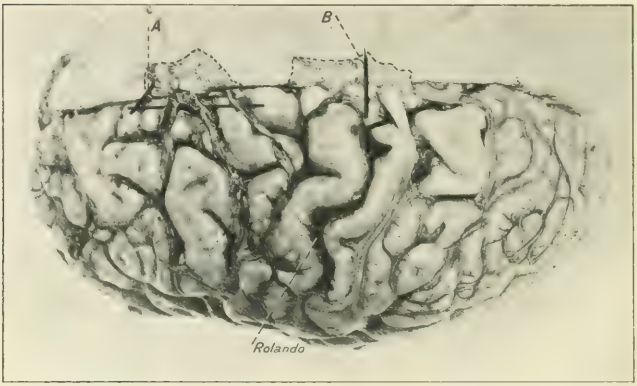

FIG. 2.—Photograph of the left side of a brain with the dura mater thrown back over to the right to show its inferior surface and the lateral surface of the left hemisphere. The frontal and parietal lacunæ are surrounded by broken lines; the entry of the larger cerebral veins into their under surfaces is clearly seen. Those entering the frontal lacuna as well as the smaller vein which passes from the precentral sulcus into the parietal lacuna are thrombosed, but the main post-central veins escape. The dura mater was not torn, but the sinus was compressed between the points *A* and *B* by a depressed fracture of the vault. For the sake of clearness the smaller veins which entered the sinus or lacunæ have not been preserved in this dissection, or in that from which fig. 3. is taken.

of Weber's compass points was much disturbed in the legs and in the right arm; the two points could be distinguished one centimetre apart on the left palm, while on the right they could not be recognized at double this distance, or on the soles when separated to ten centimetres.

During the time he remained in the Base Hospital he had slight difficulty in passing urine and occasionally incontinence; this he explained as due to the fact he could "only hold his water for five minutes or so," and that it then passed involuntarily if he did not receive a urinal. He was evacuated to England five weeks after receiving the wound and had

gradually improved during this time. His subsequent history is not at present known.

In other cases we have been able to keep the patients longer under observation and in some instances learn of their subsequent course. From our own observation we are of opinion that when the brain itself has not been at the same time damaged by the missile, the symptoms due to obstruction of the venous circulation diminish gradually, and will eventually disappear, almost or entirely, and this impression is borne out by the later histories we have received of patients transferred to England. The degree and rate of improvement may depend as much on the inconstant anatomical arrangement of the veins and the amount of anastomosis between the two lateral venous systems as on the severity of the lesion.

Such relatively rapid improvement in a severe case may be illustrated by:—

Case 2.—Lance-Corporal S. was wounded by a bullet at short range on December 21, 1914. He was unconscious for a short time and was afterwards unable to move either leg or his right arm. Speech was, however, unaffected, and he had only slight headache.

There was a sagittal gutter wound of the scalp six centimetres long, its anterior end slightly to the left of the midline and vertically above the tip of the mastoid and its posterior end on the midline, with fracture of the outer table, and probably depression of the inner table.

When he entered the Base Hospital two days after receiving the wound his face and tongue were unaffected, but the right arm was completely paralysed. The power of his left arm was unaffected but there was considerable sensory ataxia in its movements. The lower limbs were also powerless, and the right especially was rigid. All the deep reflexes were exaggerated, stimulation of the soles gave extensor responses and the abdominal reflexes were absent. The sense of position and the discrimination of compass points were lost in the right arm and in both lower limbs, but tactile and painful stimuli were normally appreciated everywhere.

He was occasionally incontinent, which he attributed to the fact that often he did not feel when he should pass urine.

Five days after the wound there was some return of power in the right fingers, wrist and elbow, but the movements he could execute were weak. His legs were still rigid and motionless. A week later all movements of the right arm were possible and those of the distal segments almost quite strong, but the limb was very ataxic owing to severe disturbance of sensation in it. His legs were less rigid and he was now able to flex and extend both hips, but no movement of the knees, ankles or toes was possible. The sense of position, the appreciation of passive

movement, the discrimination of compass points, and the localization of tactile stimuli were seriously disturbed in his right arm and in both lower limbs; but light touches, painful stimuli and vibration were normally appreciated. The recognition of form (stereognosis) was also defective, but not quite lost, in the right hand.

Five weeks after being wounded his right arm was slightly weak only at the shoulder, but was still ataxic, and he could now execute all movements at the hips and knees, but they were weaker than normal; the ankle and toe movements were still completely paralysed. The deep reflexes were still much exaggerated, and ankle clonus was present on both sides as well as the Babinski sign.

The rapid improvement of his symptoms continued till he was evacuated to England two months after being wounded. His right arm was then quite strong and all movements of both lower limbs were possible, though the distal segments were still slightly weaker than normal. His legs could now bear his weight, but he needed assistance in walking owing to the marked ataxia of these limbs, which was due to the sensory disturbances which, though less pronounced, were still considerable.

Before we discuss the symptoms in this large group of cases it will be advisable to consider the pathological changes to which they are due. We have been able to study these pathological lesions in a certain number of cases by post-mortem examination and have obtained microscopical preparations of the affected areas in a few, but the opportunity for a complete histological examination has not yet occurred.

We have already stated that in many of the cases in which the chief symptoms were due to injury of the longitudinal sinus or its tributary veins, associated lesions of the brain existed, and in describing the essential pathological changes it will obviously be necessary to separate such cases from those in which the venous system only has been directly damaged.

The most common type of injury is a gutter or tangential wound at the middle line of the head, which may be either sagittal, coronal or oblique. In many cases the skull, though exposed, showed no evidence of fracture of the outer table either to inspection or an X-ray examination, but the latter generally revealed a depression of the inner table at or near the middle line. In other patients both outer and inner tables were depressed and if the damaged bone was removed it was seen to present a spoon-shaped depression in the skull, the inner table being more depressed than would be expected from a superficial examination. Such depressions as a rule merely compressed the sinus or its lacunæ

and rarely injured their walls. Frequently however the injury was a perforating wound or an in-and-out wound of the skull, the entrance and exit being on opposite sides of the middle line and close to it, with considerable comminution of the bone between them ; here the sinus was generally lacerated and in certain instances it was completely cut through by the missile, but it was occasionally only compressed by in-driven fragments of bone.

On removing the skull-cap, or on operation when this was attempted, a thrombus was usually found in the longitudinal sinus, its character depending on the duration of the case, but as the sinus is divided up by irregular transverse trabeculæ, and held open by its rigid walls and the support it receives from the dura mater, it is probable that in many cases the thrombus did not occupy its whole lumen. When the injury lies to one side of the middle line the thrombosis may be limited to the lateral lacuna beneath it.

When the dura mater was removed the most striking feature was the condition of those superficial cortical veins which enter the sinus at the position of the wound ; these were swollen, firm to touch and could not be emptied by pressure, and there frequently seemed to be congestion of the neighbouring veins which were not actually thrombosed. The superior parts of the hemispheres which are drained by these veins were usually swollen and the convolutions flattened by pressure against the inner surface of the skull and generally firm to touch. On section there was obviously much œdema of the cortex and subcortical white matter and minute hæmorrhages, which were grouped more closely in the neighbourhood of the wound, were found. In a few instances there was an actual softening near the mesial fissure, the disintegrated brain matter being blood-stained and punctiform hæmorrhages were found in the cerebral tissue around it.

In a few of the cases which came to post-mortem examination and in others in which the condition could be observed during operation there were widespread subdural hæmorrhages which usually formed a thin layer of blood over the convexity, but sometimes extended to the base of the same hemisphere; in other cases cerebrospinal fluid removed by lumbar puncture was either blood-stained or straw-coloured. These superficial hæmorrhages undoubtedly produced some rise of intracranial pressure and contributed to the severe headache with which many of these patients suffered, but they seemed to play a subordinate part in the production of the other symptoms. Thin layers of hæmorrhage into the soft membranes and even sub-pial hæmorrhages are common.

Under the microscope the most striking change is the œdema of the affected areas, which is more prominent in the white than in the grey matter. Many of the superficial veins may be thrombosed and the rest are much congested. Minute hæmorrhages occur in both the grey and white matter, but chiefly in the proximity of the wound; many are mere perivascular extravasations. Occasionally slight perivascular round cell infiltrations are met with, especially in the neighbourhood of softenings. The walls of the latter consist of disintegrated tissue and large numbers of granule cells.

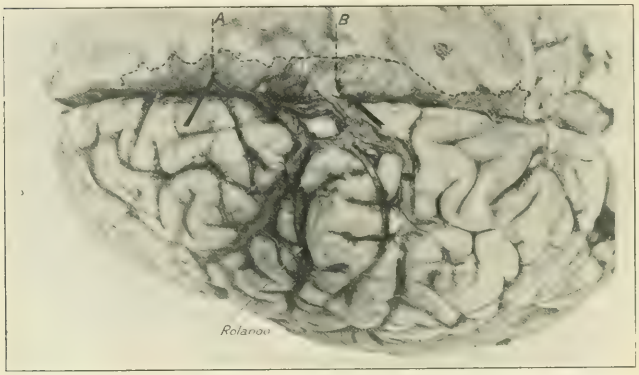

FIG. 3.—Photograph taken as in fig. 2. In this case the frontal and parietal lacunæ were not definitely separated and are both enclosed by a broken line. The longitudinal sinus and the lacunæ were compressed by a depressed fracture between *A* and *B*, and the veins which enter it at this region are completely thrombosed. The swollen, œdematous condition of the brain in the region of the thrombosed veins is obvious.

The nerve cells in the affected areas show pronounced changes; they are generally swollen and in advanced chromatolysis, the Nissl bodies having disappeared or being represented merely by irregular clumps at the periphery of the cell. Many cells, however, appear almost homogeneous and hyaline, and in relation to the age of the patient often contain an access of pigment when death did not occur soon after the infliction of the wound.

When we look at the clinical symptoms we are at first most struck by the unusual distribution and type of the motor paralysis.

The extent of the palsy naturally varies according to the site, severity and extent of the lesion; we at present have notes of 20 cases in which all limbs were affected; in 31 both legs and one arm were weak; in 16 only the lower limbs were affected; in 6 the symptoms were mainly hemiplegic, and in 5 one leg alone presented any palsy.

The distribution of the paralysis and its relative severity in different segments of the limbs is, however, peculiar, and differs from that of the cerebral palsies most commonly seen in civil practice. As in Case 1, when the upper limbs are affected the finger movements either escape or are weak for only a short time after the injury, and rapidly recover and regain their normal power. The hand movements have never remained long weak, except when the sinus condition has been complicated by an independent injury of the brain. The wrist movements, and especially those of the elbows, are affected more severely and recover less rapidly, while those of the shoulder often suffer when the more distal segments of the limbs escape, and recover much less quickly when the whole limb has been involved.

It is consequently the more proximal segments of the upper limbs which are most seriously paralysed, and the weakness diminishes distalwards. In this respect the paralysis contrasts strongly with that seen in the ordinary hemiplegia due to vascular lesions in which the distal segments of the upper limbs are almost invariably more severely affected than the proximal, and recover less rapidly. Further, a definite paresis of the face or tongue is extremely uncommon, and is at the most transient, and speech is never affected in the pure sinus injuries.

The distribution of the palsy in the legs is the converse of that of the arms; here it is always the distal movements that suffer more severely, and in slight cases and during the recovery of more severe ones we have repeatedly seen complete paralysis of the toes and ankle only, with the knee movements only relatively weak and those of the hips strong.

This distribution of the paralysis and its relative severity in the the different segments of the limbs obviously depends upon the arrangement of the cortical motor centres and on that of the cortical veins. The motor centres for the lower limbs are arranged from above downwards on the apex and lateral surface of the hemisphere in the following order—movements of the toes, ankle, knee, hip; then the small motor centres for the trunk movements intervene, and below them come in succession the motor centres

for the shoulder, elbow, wrist, and fingers. On the other hand, the superior cerebral veins which open into the longitudinal sinus drain the central gyri, as a rule, to just below the inferior genu of the fissure of Rolando, which corresponds approximately to the centre for the wrist movements, and if the area they drain be put out of function, a complete paralysis of the voluntary movements of the opposite lower limb, shoulder, and elbow, with weakness of the wrist movements, and little disturbance of those of the fingers might be expected.

The distribution of the lateral cerebral veins, and the amount and the freedom of the anastomosis between the superior and the inferior systems apparently varies greatly; and this will naturally influence the extent of the paralysis even though there be complete occlusion of the superior system; but if the lesion is slight and the occlusion is incomplete, the circulation through the smaller and more slender veins that drain the apex of the hemisphere and its mesial aspect would be more liable to become blocked than that of the larger and more thick-walled vessels; in this case only the motor centres for the more distal segments of the lower limb may suffer.

Further, while a mesial lesion is liable to produce bilateral symptoms, a one-sided paralysis of the same type may result from an injury to the one side of the middle line which blocks the circulation through a lateral lacuna, or in the veins as they enter the sinus.

When these symptoms which we attribute to venous lesions, and especially weakness of both the lower limbs, result from an injury of the vertex of the skull in the neighbourhood of the upper ends of the fissure of Rolando, they might be attributed to a direct damage or to compression of the motor centres, which lie under the wound. But in the first place the type of the paralysis, and especially the rigidity associated with it, is unlike that which occurs when other parts of the motor cortex are injured or compressed; and in the second, as in Case I, the paralysis is often too extensive in relation to the severity of the wound to permit the assumption of a direct injury. Thirdly, as the lateral parietal lacunæ overlie the more mesially situated motor centres, they must obviously be involved by any injury that would directly damage or compress them.

Even more striking than the unusual distribution of the paralysis is the rigidity which is almost always associated with it. It is generally co-extensive with the paralysis, and closely related

to it in its degree. Thus it is always most pronounced in the lower limbs, and when the upper are also involved it is greater at the shoulder than at the elbow, and is rarely present and never pronounced in the wrist or fingers.

The early onset of this rigidity is another striking point; we have seen it well marked within twenty-four hours of the infliction of the wound, and in some instances, at least, it has been noticed by the patient almost at once. It has shown no tendency to increase after the patients have reached the base hospitals, that is usually within forty-eight hours, but, on the other hand, it gradually

Fig. 4.—A sagittal section of a brain to show the multiple small hæmorrhages into the affected region, a subarachnoid hæmorrhage on its surface, and œdema of both grey and white matter extending backwards behind the fissure of Rolando.

diminishes *pari passu* with any return of power that may occur. The rigid limbs generally assume very characteristic attitudes; when the arms are affected, they lie closely adducted to the sides and rotated inwards, with the elbows flexed and pronated, and in severe cases the wrists and fingers in moderate flexion. Even the trunk muscles may be involved, and then the abdominal wall is unnaturally rigid and respiration is mainly thoracic; in a few cases, indeed, there has been slight difficulty in coughing, and phonation has been monotonous and toneless owing to the poor inspiratory intake. As a rule, the lower extremities lie fully extended at all

joints, firmly adducted and rotated inwards with the feet occasionally crossed; in fact the attitude is practically identical with that which is so characteristic of a severe cerebral diplegia. In certain cases, however, the knees are partially flexed, but they are always adducted, and rotated inwards.

The rigidity is often so great that the resistance to passive movement is extreme; it may, for instance, be quite impossible for a man of moderate strength to separate the knees. If passive movement is possible it is found that the rigidity involves all groups of muscles and is more or less equal whether the limb is passively flexed or extended. It is also continuous throughout the whole range of any passive movement that is made, and it has never shown any tendency to the "clasp-knife" type.

We have also observed that peripheral stimulation, as pricking the sole or palm, increases the rigidity, and the legs may become more rigid when the patient coughs or attempts any strong voluntary movement with his arms.

It is interesting and important that despite the great rigidity there seems to be very little tendency for contractures, that is, organic shortening of the muscles, to develop; in one severe case, of which, through the kindness of Dr. Head, we have heard eight months after the infliction of the wound, no contractures have occurred, although there was for a considerable time great rigidity of the legs.

Reflex spasms of the lower limbs have been associated with the rigidity in a certain number of cases; they have been of the flexor type, and in one case at least occurred from the day of the infliction of the wound. They may be so severe as to cause considerable discomfort to the patient, but gradually diminish in frequency and severity as improvement sets in. Reflex withdrawal of the legs can be easily evolved by peripheral stimulation, especially of the soles, but in only one case have we a definite record of an associated contralateral extension of the opposite limb.

In the rigid cases the tendon-jerks have been much exaggerated from the earliest moment at which they have come under observation; a striking feature as in ordinary hemiplegia and in cerebral palsies directly due to gun-shot injuries of the head, these reflexes are frequently absent for some time; but when the limbs are in rigid extension the extensor reflexes, i.e., the knee and ankle jerks —are much brisker than the hamstring jerks, while if in flexion the latter jerk has been the more exaggerated. Similarly in a case in which the elbows were rigid in flexion the flexor reflex, that is,

the biceps-jerk—was much increased, but the extensor reflex—the triceps-jerk—could not be obtained.

The great toes are usually permanently extended and a typical extensor response is usually obtained on stimulation of the soles, but in a few cases there has been a definite flexor response, although there was unquestionably motor paralysis of the distal segments of the lower limbs. Naturally this is also obtained in another type to which we shall later refer, in which the inability to move the limbs is due to sensory disturbance only.

Perhaps the most interesting physiological problem presented by a study of these cases is this extreme muscular hypertonus which appears very early after the injury and is closely related to the paralysis of voluntary movement. It is obviously impossible to enter here into the complex problem of the pathogenesis of rigidity, but it is now generally assumed that, as Hughlings Jackson originally taught, the increase of tone is due to removal of the inhibition which higher centres, and in this case the cerebral cortex, normally exert on the lower nervous mechanism which maintain tone in the muscles. In an ordinary hemiplegia due to a cortical or internal capsular lesion the affected limbs are, apart from some transient early rigidity, flaccid for a few weeks at least, and lose their tendon reflexes for a shorter period, and we have found the same condition in severe traumatic cerebral lesions. There must be consequently some essential difference in either the site or the nature of the pathological condition which produces paralysis in these cases, which we may group together under the title of "The Longitudinal Sinus Syndrome." It appears improbable that the level of the injury, that is the portion of the upper motor area involved, is the essential factor, as it is only the cell bodies and the upper portions of their axis cylinders which suffer with thrombosis of one of the cortical arteries, and we assume that that it is only the cell itself that is temporarily put out of function by a prolonged local epileptic attack, in both of which conditions the palsy is flaccid. This temporary flaccidity, which is later followed by an exaggeration of muscle tone, is attributed to the effect of shock which depresses, for a time, the activity of the lower centres that reflexly maintain tone.

If we look for any possible peculiarity in the nature of the cerebral lesion in these sinus cases, we are at once struck by the remarkable absence of evidence of such shock. This is especially seen on investigating the disturbances of sensation which they present; while in the early stage of an ordinary case of cortical hemiplegia

the sensory loss is partly due to functional disturbance produced by shock in the subcortical sensory mechanisms, in the cases we are considering here the sensory loss, even in the earliest stage, is almost invariably such as can be attributed wholly to a pure cortical lesion. It must be remembered too that the damage to the cortical cells associated with this sinus thrombosis is not complete or irrecoverable, the nature of the histological changes and the fact that a remarkable degree of recovery of function may occur is evidence of this, but it is improbable that the type of cell change produced by the œdema and ischæmia of the brain can be the explanation of this early persistent rigidity. On the other hand we are probably correct in assuming that the venous thrombosis produces a pure cortical paralysis unaccompanied by any shock effect on the subcortical centres which subserve muscle tone.

The sensory disturbances in these cases are especially interesting as they are almost always those of a pure cortical lesion unaccompanied by any shock effect. The appreciation of pain and temperature is unaffected and there is no definite diminution of tactile sensibility, but a certain number of light contacts are not recognized; there is, however, no threshold alteration and the proportion of those missed is not directly related to the intensity of the stimulus. On the other hand the localization of tactile stimuli, the recognition of the position and of passive movements of the limbs, as well as of form, shape and size, and the discrimination of the compass points may be seriously disturbed. The slightness of the affection of cutaneous sensibility has been frequently astonishing, as many patients have complained spontaneously of numbness or of having "no feeling" in their legs.

When the wound has been some distance behind the upper end of the fissures of Rolando, sensory symptoms have been the most prominent feature. One man, in whom there was a superficial coronal wound of the skull six centimetres behind the midpoint, had no demonstrable weakness of his legs or change in their reflexes, but he complained that both legs were numb and on examination profound loss of the sense of position and in the discrimination of compass points was found. Owing to this sensory loss he was unable to walk and on trying to do so only staggered and fell about the room; Romberg's sign was also well marked.

It is of course known that when the sense of position and the appreciation of movement are suddenly and completely abolished in a limb, aimless, involuntary movements of it may occur spontaneously. This was well illustrated by one case in

which an oblique tangential wound crossed the midline seven centimetres behind the midpoint. It is possible that the brain was directly damaged by indriven fragments of bone, but more probable that the symptoms were due to venous thrombosis.

There was no weakness or rigidity of his legs though both were very ataxic and the reflexes were normal, but in both there was pronounced sensory loss of the cortical type. When his legs were uncovered both were jerked about at irregular intervals in a curious aimless and irregular manner. Sometimes the one was raised from the bed and either thrown across or separated from the other; at other times it was quickly drawn up and extended again, or the foot was dorsiflexed or the toes moved about. The patient became conscious of the movements only when one leg touched the other or when it fell to the bed. In their impulsive aimless and incoordinate character these movements were very similar to those of chorea.

In a certain number of cases the functions of the bladder were affected. In the majority of these there was at first some difficulty in passing urine, or even retention necessitating the use of a catheter, in one case for as long as five days, but this symptom always disappeared rapidly. Less frequently incontinence occurred and in a few patients persisted for a considerable time; the bladder apparently emptied itself reflexly owing to deficient cerebral control when it had filled to a certain point, and some patients, as Case I., explained that they were able to hold their water for a few moments only after the desire to micturate had come.

As a rule the functions of the cranial nerves were unaffected, but in several patients the ocular movements were disturbed. In one group there was either temporary weakness or paralysis of the associated conjugate movements of the eyes without ptosis or affection of the pupils; one patient in whom all four limbs were affected was unable to move his eyes to order in any direction except slightly downwards, but he could follow, though not fully, a finger which was moved to either side or upwards. The visual axes always remained parallel. Within a fortnight however all movements had returned and only upward deviation was at all defective. There was a similar inability to perform all conjugate ocular movements in another patient in whom all four limbs, excepting the fingers, were paralysed and this persisted till his death on the fifth day after the infliction of the wound. More commonly, however, there was only weakness of the lateral conjugate movement of the eyes to one or both sides, or much effort was needed on the part of the patient to perform them.

We have not yet been able to make the histological examinations necessary to determine the cause of this palsy of the conjugate movements, but it seems probable that it is due to a temporary paralysis of the centres for ocular movements in the posterior part of the second frontal convolutions. We have occasionally observed a similar defect in local lesions of this region.

In other cases, eight in all, we found an isolated palsy of one or other oculomotor nerves, generally of the third, or of the third and fourth cranial nerves. This proportion is very striking when we consider the comparative rarity of ocular palsies in other types of gunshot wounds of the head; it seems probable therefore it is related to the lesion we are considering. On the other hand it might be due to a fracture of the base of the skull, to basal meningitis or hæmorrhage, or to the effect of the considerable rise of intracranial pressure which is so often present in these cases. In some cases, however, in which a post-mortem examination was made we could exclude basal lesions, and in only two of the eight cases was the sixth nerve affected, although it is of course known that this nerve is much the most liable to suffer from a pathological increase of intracranial pressure.

Fits were observed in ten of the patients; in two both sides of the body were involved, but they were limited to one side in the others. From the descriptions we received, as well as from our personal observations, the unilateral seizures apparently commenced in the face or hand when there was extensive palsy of the convulsed side, or in the lower limb when the paralysis was limited to its distal segments, that is the excitation started either in motor centres which were only partially damaged, or in their immediate proximity.

Other complications are relatively rare. Meningitis occurred in some in which the dura mater had been lacerated and the brain damaged directly; and in one a general pyæmia, from which, however, the patient recovered, developed secondary to a septic wound of the sinus. It is surprising that we have encountered only this one case of general infection, considering how commonly pyæmia occurs in connection with septic bone related to the lateral sinus. A secondary hemiplegia occurred in two cases, in one after an operation in which the longitudinal sinus bled freely and had been plugged with gauze, and in a second thirteen days after the infliction of the wound and ten days after an area of depressed bone compressing the sinus had been removed.

The general symptoms of intracranial pressure have been as a

rule pronounced. Most of the patients have suffered considerably from headache, and in some it has been particularly severe. In five cases there was also definite optic neuritis with considerable swelling of the discs, and not merely such congestion and blurring of their edges as is seen in a large proportion of all gunshot wounds of the head. In four of these cases at least, we could exclude meningitis and secondary cerebral abscess, and must consequently attribute the ophthalmoscopic change to the œdema and swelling of the brain.

The treatment of injuries of the longitudinal sinus presents considerable difficulties. When there is a defect in the skull to one side of the middle line and the brain is lacerated by indriven fragments of bone, the wound should be dealt with as if it lay in other regions of the head, but special care is necessary to avoid and control the serious hæmorrhage that is apt to occur from the sinus or its lacunæ.

If the symptoms are due, however, only to compression of the sinus or its lacunæ, the immediate removal of the compressing bone would at first sight appear to be the rational treatment; but experience has shown that the results of surgical interference have been extremely unsatisfactory. Among 39 cases we observed which were operated upon either by ourselves or others, 15 deaths occurred in the Base Hospitals, while only one among the 37 unoperated upon cases died before transference to England. These figures have not of course an absolute value as it was naturally the most serious cases which were on the whole selected for operation, and in 7 of the fatal ones there was in addition some direct injury of the brain. They are, however, sufficient to emphasize the danger of operation. On the other hand it must be remembered that the uncomplicated cases show a remarkable tendency to improve, probably owing to the free venous anastomosis permitting a re-establishment of the circulation.

If operation is necessary, it is advisable to remove bone all round the depressed portion and only then elevate this, for if hæmorrhage occurs the surgeon is then in a more favourable position to control it. As a rule some bleeding from either the sinus or its lacunæ occurs when the fragments of bone are removed, but it can generally be arrested by placing a piece of pericranial tissue or muscle on the laceration and keeping it in position for a short time by moderate pressure, and then carefully replacing the scalp flap over it. This method is certainly preferable to arresting the hæmorrhage by a gauze plug, as it is not so liable to produce

further thrombosis. When the sinus is completely divided or much lacerated a plug may be, however, necessary.

When there are serious symptoms of intracranial pressure a subtemporal decompression may be necessary; it was performed in a few of our patients. It is of course a safer operation than a large opening in the neighbourhood of the wound, as the latter exposes the patient to the risk of intracranial infection from the septic scalp. We have, however, found lumbar puncture, repeated frequently if necessary, sufficient to relieve the pressure symptoms in several cases.

As in one case a subdural clot was partly removed through a subtemporal decompression it might appear advisable to perform this operation if a meningeal hæmorrhage were suspected, but at the most only a small portion of a clot can be removed through a moderate craniectomy opening, and post-mortem experience has shown that there is usually only a thin layer of blood on the surface of the hemisphere, and that a considerable part of it spreads to the base of the brain when it is inaccessible to the surgeon.

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REPORT ON FIFTY-EIGHT CASES OF ACUTE NEPHRITIS OCCURRING IN SOLDIERS OF THE EXPEDITIONARY FORCE, INVESTIGATED AT ST. BARTHOLOMEW'S HOSPITAL FOR THE MEDICAL RESEARCH COMMITTEE.

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Though in general the health of the Expeditionary Force has been so good, some anxiety has been occasioned by an outbreak of cases of acute nephritis. At the request of the Director-General the Medical Research Committee made arrangements for its immediate investigation and the Committee have defrayed the special expenses connected with it. Arrangements were made for a number of these cases to be placed under my care in wards at the St. Bartholomew's Hospital section of the 1st London (City of London) General Hospital. Dr. Mackenzie Wallis and Dr. Trevan have made the chemical investigations involved. Captain F. W. Andrewes, F.R.S., Professor of Pathology in the Hospital Medical School, has reported on the morbid anatomy of the kidney, and the pathological investigations have been carried out in the pathological department of the hospital of which he is the director. Dr. R. G. Canti has particularly interested himself in carrying out many of these. Mr. Foster Moore has reported on the condition of the eyes in the majority of the cases. Without the cordial co-operation of all these gentlemen this investigation could not have been attempted.

It has been thought advisable to issue this interim report for the information of those medical officers who are engaged in treating these cases of nephritis, before the whole investigation is complete. Observations confirmatory of, additional, or opposed to those herein described would be welcomed. In this way it is hoped that a useful contribution to the medical history of the War may be arrived at.

Incidence.

It appears that 1,062 cases of acute nephritis occurred in the Expeditionary Force up to the end of June. The monthly returns show that very few cases occurred till February, in which month 72 cases were reported, 138 in March, 220 in April, 211 in May and

326 in June. Of these cases 58 have been investigated at St. Bartholomew's Hospital. These figures differ strikingly from those of the South African War. The late Dr. Washbourn reported that "acute nephritis was singularly infrequent." At Deelfontein there were only six cases of acute Bright's disease in whom the disease was contracted in South Africa. Old standing nephritis with an acute attack superadded was more frequent. Moreover, there can be little doubt that the disease has been much more frequent in this campaign than in civil practice. As acute nephritis is not a notifiable disease it is impossible to provide exact figures, but it may be said that at St. Bartholomew's Hospital, where the number of medical cases admitted in a year averages 7,000, there were in a period of nine years only 166 cases, 120 males and 46 females. In a period of five years only 26 cases were admitted in men between the ages of twenty and forty, which is a better standard of comparison. Again, among the troops serving in the United Kingdom the incidence of nephritis closely approximates to the peace ratio for this disease. We may conclude that ordinarily acute nephritis is a rare disease in men of a military age and that a special explanation must be sought for the present frequency of the disease in the Expeditionary Force.

It was thought by some observers that the condition was commoner in the older soldiers, but the ages of the patients admitted to St. Bartholomew's Hospital do not bear this out. Arranged in hemidecades the age incidence was as follows :—

Under 20	2
20 to 24	14
25 to 29	11
30 to 34	13
35 to 39	10
40 to 44	5
45 or over	3

Etiology.

In dealing with a comparatively small proportion of the total number of cases it is of not much value to tabulate the places at which the disease was contracted ; the cases came chiefly from

Ypres	15
Bethune	12
Boulogne	6
Givenchy	4
Festubert	3
Floubaix	3
Richebourg	3

Exposure to wet and cold has always been held to play an important part in the causation of acute nephritis, but this epidemic goes far to disprove that contention. For during the winter when there was much wet weather the cases were few and far between, and it was not until the weather was better that the disease assumed epidemic proportions. In each case, however, careful inquiry was made into the question of recent exposure to wet; in twenty cases there was a definite history of this, but in thirty-eight cases no history could be obtained.

The water supply has also been suggested as a possible cause; whether by reason of its being obtained from an unwholesome source, or by chlorination or from metallic poison derived from the galvanized water-carts or the water-bottles. It may be said at once that these cases lend no support to any such hypothesis; the sources from which the patients had obtained water were extremely variable, as is shown by the following table:—

Source from which water was obtained	Wholly	Partially
Water carts	17 ..	7
Pumps	13 ..	13
Dug-outs	5 ..	7
Springs	1 ..	4
Ponds	1 ..	0
Wells	1 ..	3
Town water supply	1 ..	1

Moreover in some cases the patients had been careful about boiling the water, in others they had not; in some cases the water had been chlorinated, in others not.

Another suggestion was that the disease was rife amongst the horses. We need not stop to inquire whether they derived it from the same source as the men or whether it was transmitted by or to the horses, since investigation has failed to reveal the existence of the disease among the horses to any extent. Only ten of the patients had had the handling of horses; it is interesting that one of these volunteered the statement that his horses had strangury.

None of the men admitted for acute nephritis had been wounded, and no case of acute nephritis occurred among the wounded men admitted to St. Bartholomew's Hospital. Clearly, if the disease is due to an infection, a wound infection plays no part in it. As respiratory symptoms have been common in this series of cases, it should be mentioned that only two of the men had previously suffered from gas asphyxia.

Excessive protein diet has been suggested as a possible cause.

It seems highly improbable that one hundred and eighty grammes of protein would cause acute nephritis in a number of otherwise healthy men, and that without impairing the health of the others in any way. Moreover, as will appear later, the evidence is in favour of an infective rather than a merely toxic change in the kidneys, and, a more important point, there were no signs of an intestinal toxæmia.

On this point Dr. Mackenzie Wallis's observations appear quite definite. Proteins as such would not exert a toxic influence; they could only do so through their putrefactive products. Among the more striking of these products is indican, which can be readily recognized in the urine. But it is not altogether a safe guide to the total amount of putrefactive change. The putrefactive bodies are largely combined with sulphates and excreted as ethereal sulphates; indican is merely one of these. Normally the ethereal sulphates form about one-tenth of the total sulphates. With intestinal intoxication the ethereal sulphates rise both absolutely and relatively. In the cases investigated the total amount of ethereal sulphates was low, probably because of the relatively low protein intake, and the proportion of ethereal sulphates did not show an increase. Another guide to the amount of intestinal putrefaction is the presence of urobilin in the urine. Urobilin is derived from the bile pigment in the bowel by the reducing action of the bacteria there. In this series of cases urobilinuria was not observed. If fermentative rather than putrefactive changes predominate, oxalates are apt to be formed in considerable quantities and crystals of calcium oxalate are found in the urine. In this series calcium oxalate crystals were only found twice. So that the evidence is all against excess of either putrefactive or fermentative changes in the bowel.

Acidosis has been suggested as a cause, on account of the frequency with which dyspnœa occurred in the early stages of the disease. It is not clear how acidosis could arise on the diet given to the troops, nor is there a general agreement that acidosis can cause nephritis. The cases of dyspnœa investigated did not show acidosis as a constant feature.

In a few cases the disease was evidently an exacerbation of old trouble, there being a clear history of previous attacks in 5, 3 of them being apparently scarlatinal in origin. In 3 others there was a history of scarlet fever but no evidence of previous kidney trouble.

An ætiological factor of some interest was the high percentage

of positive Wassermann reactions. The reaction was done in 56 cases and was positive in 18, being strongly so in 4, and feebly so in 3. That is to say, practically one in three of the cases was positive, and though this is too low to make a specific taint the cause of the disease, it is high enough to make it a probable factor in lowering the patient's resistance. It is noteworthy that in three out of the four instances where the reaction was strongly positive there was evidence of extensive kidney damage, with a tendency to prolonged hæmaturia and to relapse. I gather that an incidence of nearly thirty-three per cent is higher than the incidence of syphilis in the Army in general. It may be added that the majority of the positive reactions were in the long service men, though three out of the four strongly positive reactions were in men who had enlisted for this war.

Pathology.

That the disease may be considered a form of nephritis is sufficiently shown by the combination of œdema with albuminous urine which also contains blood and casts. But a nephritis may be the result either of an infection or an intoxication. Taking scarlet fever as the classical example of an acute nephritis due to an infection it is generally admitted that the stress of the disease falls especially on the glomeruli, though the tubules are not spared. In a toxic nephritis, on the other hand, such as that produced by corrosive sublimate, the lesion is tubular. It seemed important to determine how far the glomeruli and the tubules were respectively involved in these cases, as the methods of combating an infection and a toxic agent would differ widely.

Certain tests are relied upon to differentiate the parts of the kidney chiefly affected, and I may summarize here the principal results obtained by Dr. Mackenzie Wallis and by Dr. Trevan, which will be published *in extenso* later.

(1) *The Chloride Test.*—That chlorides are often badly excreted in nephritis is well recognized. In 1911 Schlayer went further and attempted to differentiate between tubular and glomerular damage by the reaction of the kidney to ingested chlorides. A kidney that is healthy can get rid of five grammes of salt by merely increasing the concentration of the urine: but if the tubules are damaged it cannot excrete urine of the requisite concentration. If the glomeruli were intact they could come to the assistance of the damaged tubules by excreting more urine of a low concentration and thus

help in the excretion of the salt. It is claimed that in an early stage of glomerular damage there is an exaggerated response on their part producing excessive diuresis, so that more salt is excreted than was administered. But if the damage to the glomeruli is more profound they are unable to respond by increasing the output of water, which may even fall. A rise in the percentage of sodium chloride in the urine, with a fall in the total quantity of this substance excreted, would be evidence that the tubules could respond while the glomeruli could not. The absence of a rise in the percentage of sodium chloride or an inadequate rise would be evidence, on the other hand, that the tubules were damaged.

This line of investigation has been carried out by Dr. Trevan in twenty-three cases. As some of these were getting rid of previously retained chlorides, i.e., were recovering from a tubular lesion, it was necessary to continue observations on the chloride output on a diet of known chloride content for some days before administering the dose of five grammes of salt. The general conclusion reached was that all the cases investigated showed damage to both glomeruli and tubules. In twelve cases, i.e., just over fifty per cent, the glomerular damage was sufficiently severe to produce an actual fall in the quantity of water excreted after the dose of salt.

He confirmed the fact of salt retention in four cases by determining that the chlorides in the blood were higher than normal.

(2) *The Iodide Test.*—According to Schlayer iodide is excreted by the tubules. In eight cases Dr. Trevan estimated the time taken to excrete a dose of two grams of potassium iodide, and also the amount excreted in certain periods of time. A great reduction in the total amount excreted was shown in every case. The evidence as to tubular damage as given by the chloride and iodide tests agreed fairly well in these eight cases. The difficulty in excreting iodide was further evidenced by the intolerance to iodide shown in those cases where it was given therapeutically because of a strongly positive Wassermann reaction.

(3) *The Lactose Test.*—Lactose injected intravenously appears to be excreted by the glomeruli. In three cases where this test was tried, Dr. Trevan obtained marked evidence of extensive glomerular damage, as there was marked delay in excretion. As a rigor occurred after the injection in one case, and hæmaturia in this and one other case, the test was discontinued.

(4) *The Diastase Test.*—Of recent years the amount of diastase

present in the urine has been used in the diagnosis both of pancreatic disease and renal permeability. In the presence of the former the diastase output is greatly increased. Given the absence of this the amount of diastase should vary between 10 and 22·2 units. But if the renal permeability is diminished this amount may fall to 5 units or less, and perhaps to 0. A colloidal substance such as a ferment would probably be excreted by the tubules. Dr. Mackenzie Wallis, who has paid special attention to this test for the last two years, has estimated the urinary diastase in fifty of these cases, in many of them on more than one occasion. The results have been interesting and valuable. In no instance was the diastase output increased, as it often is in toxic processes. In thirty-one cases the diastase value was low, and eight had none at all in the urine. Nineteen cases showed a normal value. Speaking generally, this method agreed with the others in indicating the cases with definite tubular damage. Those with normal values were usually slight cases. But further, this method was of distinct prognostic value, for those cases with a normal value generally recovered quickly, while those with a low value ran a more protracted course and showed a tendency to relapse. As the cases improved the diastase value tended to rise. Again in some cases the low diastase value persisted after the albuminuria had ceased, showing that the kidney had not completely recovered. The test has an important bearing on the prognosis, and a case of acute nephritis cannot be said to have recovered completely until the diastase value has returned to normal.

The Nature of the Proteins Present in the Urine.

Though this does not enable us to distinguish between the glomerular and tubular damage its determination enables us to distinguish an actual inflammatory lesion of the kidney from a functional albuminuria and from the so-called "leaky" kidney, which is not the seat of an inflammatory lesion. Dr. Mackenzie Wallis's investigations on this point show that the ratio of albumin to globulin in these cases ranged from five to one to six to one, which accords with all previous observations upon inflammatory conditions of the kidney.

All these tests taken together point to a nephritis of a diffuse type usually thought to be infective in origin and bearing no relation to that caused by metallic poisons.

Morbid Anatomy.

Up to the present only one of the cases at St. Bartholomew's Hospital has proved fatal, and a full report on the kidneys is not yet forthcoming. It can be said, however, that they showed extensive glomerular with some tubular change. One case, under the care of Major Calvert, at the First London General Hospital, died, and Captain F. W. Andrewes has furnished a report on the post-mortem examination and the histological appearances of the kidney. He found that all the glomeruli showed some evidence of inflammation, but that some were affected more than others. There was extensive damage in the convoluted tubules. The interstitial tissue of the kidney was œdematous and infiltrated with lymphocytes and polymorphs. The change was apparently recent, for there was no fibrosis. No micro-organisms could be found in stained sections. He concluded that the appearances were those of a fairly recent subacute diffuse nephritis, and that there was nothing to suggest that the nephritis was of any distinct or unusual type.

There is therefore a substantial agreement between all the methods of investigation. The improbability of a simple toxic agent or an intestinal toxæmia has already been shown.

We must next consider the steps that have been taken to detect an infective agency.

(*a*) *Blood Cultures.*—These were made in three cases, in two because of serious symptoms of a possibly septicæmic nature, and in the other because it was admitted to the hospital in an early stage of the disease. In all cases the blood was sterile.

(*b*) *Cultures from the Urine.*—Catheter specimens were obtained in twenty-one cases and cultures made. In eighteen of these the urine proved to be sterile. In the remaining three, *Bacillus coli communis* grew, but in two of these only when one cubic centimetre of the urine was taken, while the remaining case was not a typical one being much more of the type of pyelitis, which came on during an attack of typhoid fever.

There is therefore no evidence of the nephritis having been set up by an ascending infection, or by a blood infection, the stress of which fell upon the kidney. This accords with Captain Andrewes's observations on the morbid anatomy of the kidney.

(*c*) *Throat Cultures.*—Seventeen of the cases complained of a sore throat as an early, if not the initial, symptom. In view of the generally accepted association between tonsillitis and nephritis (see Sir Wilmot Herringham's "Kidney Diesase") it seemed important

to investigate this point. Cultures were taken from all the throats where soreness was complained of, and in several in which no such complaint had been made. It was found in every instance that streptococci grew in abundance, and sometimes in practically pure culture. Pneumococci, diphtheroids and *Micrococcus catarrhalis* were also found in some cases. Before it could be concluded that the streptococci were responsible in any way it was necessary that control observations should be made, since streptococci are normal inhabitants of the throat. Dr. R. G. Canti therefore made cultures from the throats of ten soldiers with nephritis, ten wounded soldiers who had not got nephritis, and ten civilian patients suffering from various surgical diseases. His report shows that the throats of the soldiers were, as far as bacteriological examination went, in a healthier condition than the civilians, while the throats of the soldiers with and without nephritis were closely similar. He is engaged in estimating the streptococcal antibodies in the blood of some of these cases. This would afford evidence of a recent streptococcal infection. So fart here is no proof of the presence of any excess of these antibodies.

While this points away from a possible streptococcal origin, there remains the possibility of a filter-passing ultramicroscopic organism being the cause. The comparative frequency of sore throat as a symptom and the failure to find a bacterial cause for a disease which is almost certainly of infective origin make it very important that a filter-passer should be looked for. Animal experiments with this object in view are now in progress, but it is too early to speak of results yet. It has been suggested that the cases are due to a suppressed form of scarlet fever, which is also thought to be due to a filter-passer. It is interesting in this connexion to note that so far cases of nephritis have not occurred among the Indian troops, and the natives of India are said to enjoy comparative immunity from scarlet fever. It is also interesting, in view of the frequency of otitis media in scarlet fever to note this occurred in two cases just before the œdema. But as the ordinary incidence of nephritis in scarlet fever is ten to twenty per cent, this view would entail the conclusion that there had been ten to twenty thousand cases of scarlet fever had occurred among the troops up to the end of June. The general health of the troops has been so good as to make such a large number of suppressed cases unlikely. Moreover, even in "scarlatina *sine eruptione*" desquamation follows, and this has not been observed in these cases.

Signs and Symptoms.

In the majority of cases the first thing noticed was the œdema, which usually started in the face and legs, and was curiously localized in some cases. The swelling became generalized in most cases. In one case admitted on the second day of the disease the legs and scrotum were very œdematous, but the rest of the body escaped. Œdema was almost a constant feature, being present in 53 cases, and of the 5 in whom it did not occur 4 gave a history of previous nephritis. In every case it soon cleared up.

Pain in the back was common at the outset, and was sometimes the initial symptom. It was not so common as œdema, however, being experienced in 37 cases. "Biliousness," nausea, vomiting and abdominal pain were given as initial symptoms by 6 patients, while 2 first complained of pains all over. Cough, diarrhœa and headache were occasionally the initial symptoms. Two cases developed an aural discharge just before the onset of the nephritis, again pointing to a possible throat infection. As already stated, 17 patients complained of sore throat at the beginning.

One of the most striking points was the frequency with which shortness of breath occurred as an early symptom. It was present in 49 cases, and only absent in 9. Of these 9, 2 had no œdema, while the œdema was slight in 2 others. Œdema occurred without shortness of breath in 3 cases. As a rule this shortness of breath started at the same time as the œdema, but did not last so long, having ceased at the end of two or three days. Only 5 cases had any dyspnœa while in St. Bartholomew's Hospital. The alveolar CO_2 was determined in each of these by Dr. Trevan: in 3 it was normal, while in the remaining 2 it was reduced from the normal five per cent to about three per cent. Dyspnœa due to acidosis is always associated with this reduction of alveolar CO_2 and uræmic dyspnœa is of this type. In three of the cases it would therefore appear that the dyspnœa was not uræmic.

Dyspnœa is not regarded as a common feature of acute nephritis. It may occur from uræmia or cardiac failure, of course, but the majority of these patients were not so ill that either the kidney or the heart was failing, and none of them showed signs of cardiac dilatation. The close agreement between the onset of œdema and dyspnœa suggests that the outpouring of fluid into the lungs or pleural cavities may have been responsible for the shortness of breath. This is supported by the frequency with which a cough

and bronchitis also occurred. Some of the early cases showed well-marked respiratory distress after their journey from France.

A slight and irregular temperature was common in the earlier stages and was sometimes prolonged where recovery was slow. More or less severe headache was common and was sometimes persistent. Anæmia was present in a fair number of cases, especially in those with a positive Wassermann reaction.

The urine was noted by the patients to be altered in most cases, though 11 failed to observe any change, 18 noted that it was scanty, while 8 observed that it was increased in amount. One patient noted great variation in the amount he passed; 8 had increased frequency of micturition, 3 had strangury, and 2 had incontinence; 22 observed that the urine was dark or actually contained blood.

While in hospital the variations in the quantity of urine were often striking, as illustrated by the following five cases, in which the extreme limits are given:—

52 to 140 oz.
60 ,, 160 ,,
30 ,, 125 ,,
35 ,, 108 ,,
42 ,, 128 ,,

The amount of urine was definitely reduced in fourteen cases; in these the output varied between eleven and thirty-seven ounces. But such extreme reductions as are usually seen in acute nephritis were not observed, and most of the above soon excreted larger quantities, while in some the amount was always increased. The highest figures observed were:—

100 to 120 oz. in	..	..	8 cases
120 ,, 140 ,,	..	..	5 ,,
140 ,, 160 ,,	..	..	2 ,,

This variability in amount with frequently marked increase is due to what Muller called hyposthenuria; the kidney being unable to excrete a concentrated urine. Herrick says that it occurs not only in chronic interstitial nephritis, but also in acuter forms, especially when there is glomerular involvement. It was certainly a more marked feature of these cases than in the acute nephritis met with in civil practice.

The amount of albumin differed greatly. Usually it varied from a trace to 0·3 per cent as estimated by Esbach's method, but higher readings, such as 0·5 to 0·8 per cent were obtained occasionally. Some were estimated by the more exact Aufrecht method and yielded readings ranging from 0·001 to 0·3 per cent.

Red blood corpuscles were found in forty-four of the fifty-eight cases. The amount was very variable. Sometimes the urine was bright red with blood, sometimes merely smoky, and often chemical and microscopical tests were needed to detect the blood. A common feature was the settlement of a flocculent reddish-brown precipitate in which alone the blood could be found.

The reaction with tincture of guaiacum and ozonic ether was not found to be a reliable test for the presence of blood.

White blood corpuscles were present in thirty-six cases; sometimes only a few were found, but usually they were more abundant than is the rule in acute nephritis. Isolated renal cells, transitional epithelial cells, squamous cells from the lower urinary tract and cells from the genital tract were commonly found also in the centrifugalized deposit.

Casts were found in all but three cases. By far the commonest thing was to find a mixture of epithelial, granular and hyaline casts. Blood casts and fatty casts, on the other hand, were uncommon. The frequency with which the different forms were found may be tabulated thus :—

Blood casts	2
Epithelial casts	36
Fatty casts	7
Granular casts	51
Hyaline casts	33

Crystals were rare in the urine; calcium oxalate crystals were found twice, and uric acid crystals once. The rarity of organisms in catheter specimens has already been referred to. *Bacillus coli* was found in only three cases; in only one were they abundant, and this case also had pyelitis after enteric fever.

The systolic-blood pressure was estimated in every case, and usually on several occasions. The figures obtained were not very helpful; perhaps at the outset the pressure would have been found to be raised in all cases; it was one hundred and fifty-five in the case admitted on the second day of his disease. The figures obtained as soon as the patient had settled down after his journey may be tabulated thus :—

Blood-pressures	Cases
Under 120	4
120 to 129	13
130 to 139	10
140 to 149	10
150 to 159	9
160 to 169	6
170 to 179	3
180 and over	3

It may be said in general that as the patient improved, the pressure tended to fall to normal. The cases with a history of former attacks gave readings of 130, 148, 152, 155, and 200 respectively, but in a few days the 130 had fallen to 110, and the 200 to 140.

It cannot be said that the blood-pressure had a definite prognostic value. Thus three men were admitted with a pressure of one hundred and twenty-eight. The first had reached the eleventh day of his illness; eight weeks later there were still red corpuscles in his urine. The second was at the seventh day of his disease; ten weeks later he still had hæmaturia. The third had been ill a month, and he died eight weeks after admission. On the other hand, cases admitted with blood-pressures of 165, 168, and 179 have proved obstinate. Probably the best thing is a moderately raised pressure at the outset, falling fairly quickly to normal. Such cases have usually made a good recovery.

The eyes were examined by Mr. Foster Moore in fifty cases. He found—

No changes	37
Retinal hæmorrhages	4
Old iritis	1
Corneal nebula	1
Subconjunctival hæmorrhages	2
Conjunctivitis	1
Choroiditis	2

Of these changes only the retinal hæmorrhages were definitely associated with the nephritis, though the subconjunctival hæmorrhages were probably due to high pressure.

Three patients had fits which were presumably uræmic in origin. One of these had been venesected for fits in Boulogne. The other had a severe fit after reaching England and bit his tongue very badly. He had another epileptiform fit in the ward; yet he had never had fits before or after these two. His Wassermann reaction was negative, and he made a good recovery.[1] These were the only complications observed.

It may be added that the milkiness of the blood serum referred to by Bright in his original account of the disease was not observed in any of the fifty-six cases in which the blood was drawn for

[1] Since writing this, one new case had violent uræmic fits immediately after admission. They quickly yielded to free venesection.

Wassermann's reaction, but was present in the fatal case under Major Calvert's care.

Course and Prognosis.

On the whole it may be said that the disease has run a favourable course. Only one of the fifty-eight cases has died, and in no other case has life appeared to be in danger. It may also be assumed that the cases sent home have been the more protracted ones. The patient who developed the disease immediately after his return from France on leave, and who was admitted to the hospital on the second day of the disease, was free from albuminuria on the fourth day. Most of the cases have felt quite well as soon as the œdema subsided, though some have complained of headache, cough and digestive disturbances for a week or more. Yet many of those who felt and looked quite well were still passing albumin, blood and casts. A curious feature has been the tendency to relapse, in that there has been a return of albuminuria and hæmaturia, generally without ascertainable cause, though in some cases the first motor drive might be held responsible. The fact that they had been permitted to go for a drive shows that they were considered to be convalescent at the time.

Up to the present (August 5), twenty-two have apparently recovered. A considerable number of the remainder are still under treatment. Final statistics are not yet forthcoming, but the following data may prove of interest.

Blood was never found in fourteen cases. It disappeared from the urine in

10 days in	..	..	..	1 case
3 weeks in	..	..	..	2 cases
4 ,,	..	..	..	3 ,,
5 ,,	..	..	..	2 ,,
8 ,,	..	..	..	3 ,,
9 ,,	..	..	..	2 ,,

It was still present at the end of

6 weeks in	..	..	..	2 cases
8 ,,	..	..	..	4 ,,
9 ,,	..	..	..	2 ,,
10 ,,	..	..	..	2 ,,
12 ,,	..	..	..	3 ,,
14 ,,	..	..	..	1 ,,
20 ,,	..	..	..	1 ,,

Albumin disappeared from the urine in

4	days in	1 case
10	,,	1 ,,
2	weeks in	1 ,,
3	,,	4 cases
4	,,	3 ,,
5	,,	3 ,,
6	,,	1 ,,
7	,,	1 ,,
8	,,	3 ,,
9	,,	2 ,,
10	,,	1 ,,
14	,,	1 ,,

It was still present at the end of

6	weeks in	4 cases
7	,,	4 ,,
8	,,	7 ,,
9	,,	4 ,,
10	,,	2 ,,
11	,,	1 ,,
12	,,	5 ,,
14	,,	1 ,,
15	,,	1 fatal case
20	,,	1 case

Casts have been observed to disappear from the urine at the end of

4	weeks in	2 cases
5	,,	2 ,,
12	,,	1 ,,
16	,,	1 ,,

It has generally been observed in this series of cases, that as improvement occurred the units of diastase present in the urine rose to a normal level. Comparing the diastase reaction with the time of disappearance of the casts, it may be noted that in one case where the casts went in four weeks the diastase reaction was normal, that in another where they went within the same time, the diastase rose to normal as the casts disappeared. Where the casts disappeared in five weeks the diastase steadily rose to normal. On the other hand, where three months elapsed before the casts disappeared the diastase remained very low, and where four months were needed for the casts to go the diastase remained low until about the time this happened. But casts vanished in five weeks in one case where diastase was entirely absent, and it is interesting to note that this man still felt ill; he was one that had a strongly positive Wassermann reaction. In several instances there seemed to be no agreement between a normal diastase reaction and the

cessation of hæmaturia, but in general it may be said that a normal diastase reaction is of distinctly favourable prognosis, especially in the absence of hæmaturia.

All these dates have been calculated from the onset of symptoms and not from admission to St. Bartholomew's Hospital.

The fatal case suffered throughout from much anorexia, repeated vomiting and irregular temperature. Blood cultures were made on two occasions as a secondary infection was suspected; no growth was obtained, however. He developed more bronchitis, and a doubtful pericardial friction was heard on two days. He gradually became weaker, and passed less urine (on one day only eight ounces) and died at the end of fifteen weeks from the onset of symptoms. He never had any fits; the blood-pressure was only 128, and the Wassermann reaction was positive. Unfortunately, no post-mortem examination was allowed beyond the removal of the kidneys, which will be reported on later.

Treatment.

There is unfortunately not very much to be said under this head. Speaking generally, the cases were kept in bed as long as there was any hæmaturia or even marked albuminuria. Otherwise, unless there were some special symptoms the patients were allowed up, and when convalescent went into the square. The hospital milk diet was given in the first instance. This consists of :—

Tea	1 pint
Bread	14 oz.
Milk pudding	8 ,,
Milk	2 pints
Butter	1 oz.

This diet contains approximately :—

Proteins	86 grm.
Fats	77 ,,
Carbohydrates	353 ,,
Calories	2550 ,,

As improvement occurred, the next step was to add two eggs to the diet, giving it an approximate value of :—

Proteins	100 grm.
Fats..	90 ,,
Carbohydrates	353 ,,
Calories	2,720 ,,

Cases which did not seem to do well on this were put on a special "Low Nitrogen Diet," on the view that in acute nephritis

a period of comparative nitrogen starvation would afford the most rest to the kidney. This diet was only given for a short time, though occasionally as long as a week if it was well tolerated. It consisted of :—

Bread	6 oz.
Butter	1½ ,,
Potatoes	10 ,,
Greens	4 ,,
Salad	3½ ,,
Boiled rice	8 ,,
Milk	½ pint
Stewed fruit and sugar.	

This has a value of :—

Protein	54 grm.
Fats..	50 ,,
Carbohydrates	290 ,,
Calories	1,615 ,,

The chief deficiency in this diet is the lack of fat. In some cases it was improved by the addition of some cream cheese or salad oil with the lettuce. Its advantage was that while reducing the protein intake to about Chittenden's standard, it gave the men a greater variety. The rice was sometimes cooked with a tomato flavouring, which was appreciated. It could easily have been improved by the addition of jam, but the men having had jam constantly in their rations, did not like this. Exceptionally it was improved by the addition of honey, and but for the difficulty of cooking for a number of men, the fat could have been increased by frying the potatoes in fat or mashing them with butter. In this way a diet can be constructed which permits of variety while giving the kidney little work to do. Unless a considerable amount of fat is taken, however, the diet is of low calorie value, and is not suitable for prolonged use. Another drawback is that it requires a fair amount of salt to make it palatable.

It is difficult in a disease of such variable duration to assess the value of any treatment, but my impression is that this low nitrogen diet was a distinct benefit in several cases.

Where salt retention seemed to be a prominent feature, saltless bread and butter were given. While the salt excretion was being determined, a diet of known salt content was given, with saltless bread and butter, and a known weight of salt (one or two grammes, usually the latter) was added.

Drugs.—Potassium citrate was given where the excretion of urine was scanty or painful. Stimulating diuretics were never

given except theocin in some test cases. Theocin sodium acetate belongs, of course, to the caffeine group, but is claimed to have the power of increasing the permeability of the kidney. It occasionally seems to be of definite benefit in chronic nephritis. It was tried in three of these cases. In the first case it had a marked diuretic effect, and also raised the diastase content of the urine from 0 to 20. In the other two cases it had no effect on the diastase, and only a slight diuretic action. One of these cases also had a return of hæmaturia when the theocin was given. Theocin, therefore, like caffeine, diuretin, and theobromine, has an irritant effect as well as a diuretic action. It is an unsuitable drug as long as there is any degree of acuteness of the disease; in the more chronic stages, however, it does definitely increase the permeability of the kidney. When the drug is stopped, the permeability of the kidney returns to its former level. The dose administered was two grains twice a day.

Bicarbonate of soda was given in drachm doses where there was shortness of breath, and with definite benefit when the dyspnœa was accompanied by a low alveolar CO_2.

Potassium iodide was given where the Wassermann was strongly positive, but it was not well borne, and generally had to be discontinued. In one case its administration was followed by marked œdema of the eyelids, which soon subsided when the drug was discontinued. This is, perhaps, to be expected, considering the evidence given in Dr. Trevan's report as to the difficulty with which iodide was excreted by these patients.

Several drugs were tried for the relief of hæmaturia. Emetine having been tried with some success for various forms of bleeding, such as hæmoptysis, was given a trial in several cases, but without benefit. Liquid extract of ergot was tried in a large number of cases in twenty-minim doses three times a day. It generally diminished the amount of blood for a time, but it did not appear to be responsible for bringing the hæmaturia to an end. Tincture of hamamelis was also tried, but did not seem to give as good results as ergot. As already pointed out, the duration of the hæmaturia was so variable that attempts to estimate the value of a drug in its treatment are beset with fallacies.

Conclusions.

(1) These cases are true examples of acute nephritis, as shown by (*a*) the combination of œdema with albuminous urine containing casts; (*b*) the tests for renal permeability; (*c*) the nature of the proteins in the urine; (*d*) post-mortem evidence.

(2) The cases have been too prevalent to be merely accidental, and the close similarity of the symptoms suggest a common cause.

(3) Exposure, water supply, metallic poisons, and intestinal toxæmia can be excluded as the cause. The prolonged cases showed a high percentage of positive Wassermann reactions.

(4) The curve of incidence suggests an infective agent as the cause; this is supported by the degree of affection of the glomeruli, as shown by the chemical tests and the post-mortem evidence.

(6) Bacteria can apparently be excluded as the infective agent, but a filter-passing ultra-microscopic organism cannot be excluded. The point of entrance of the infection is possibly the tonsils.

(7) The outstanding clinical features of the epidemic are: (*a*) the frequency with which shortness of breath occurred at the beginning, which was not always associated with acidosis; (*b*) the great variability in the duration; (*c*) the tendency to remission and relapse; (*d*) the ultimately favourable prognosis.

THE CAUSATION AND PREVENTION OF ENTERIC FEVER IN MILITARY SERVICE, WITH SPECIAL REFERENCE TO THE IMPORTANCE OF THE CARRIER.

Being an Account of Work done at Naini Tal Enteric Depot, 1908-11.

By Major D. HARVEY.[1]
Royal Army Medical Corps.

(*Continued from p. 508, vol. xxiv*).

Prevention.

It follows, then, that if the cause of enteric fever and paratyphoid fever is the presence of infected persons in a susceptible community the prevention of those fevers will lie in the detection and removal of such individuals.

To detect an unknown carrier, especially among a large number of inoculated men, is an exceedingly difficult task. If he should give rise to a few fresh cases his detection may be facilitated, but it is far better to go to the fountain head, and by the early detection and isolation of all enteric fever cases remove the carrier, or, at any rate, prevent his return to ordinary duty.

The writer is firmly convinced that in military service at least all carriers have previously suffered from an attack of enteric fever. It follows, then, that by the rapid and accurate diagnosis (by blood culture, Widal reaction and clinical picture) of all cases of fever, and the isolation and examination of all convalescents, it will be possible to eliminate carriers. Those who were carriers previously will automatically disappear as they become time-expired.

Such a condition of affairs now prevails in India, where we have a "carrier-free" Army.

To relate how this has been accomplished is the purpose of the remainder of this essay.

However, no paper dealing with the prevention of enteric fever in military service would be complete without a reference to anti-typhoid inoculation. The writer is well aware that but for the reduction of enteric fever brought about by inoculation it would have been impossible to deal adequately with convalescents; the

[1] Now Lieutenant-Colonel.

depots would at their inception have been overwhelmed by numbers.

Typhoid inoculation was first introduced into the Army in 1898, just prior to the outbreak of the South African War, by Professor (now Sir Almroth) Wright. He based the principles of his method on that of Haffkine for cholera. The vaccine consisted of a broth culture of typhoid bacilli of ten days' growth, killed by heating to 60° C. for half an hour. This temperature was regulated by Dr. Wright's ingenious paraffin thermometer, which indicated when a temperature of 60° C. had been attained, but, unfortunately, placed no check on its going much higher subsequently, as indeed it did frequently in the personal experience of the writer. At the time this was not thought to be a vital point and was regarded as being useful to " mac siccar."

The use of this vaccine was not attended with the happiest results, as the local and general reactions it produced were severe and the protection not always of a very high degree, although statistics collected from units during the South African War undoubtedly showed that there was a certain amount of protection, and also that if infection followed it was as a rule more mild in the inoculated than in the uninoculated.

Further investigations were conducted by Lieutenant-Colonel (now Sir William) Leishman and Major W. S. Harrison, R.A.M.C. Their object was two-fold :—

(1) To reduce the severity of the reaction.

(2) To increase the protection afforded.

The results of these investigations, which entailed a great deal of most elaborate and careful scientific work, were as follows :—

(1) A vaccine heated to 60° C., or over, for half an hour, is practically inert.[1]

(2) A vaccine which has been kept for more than three months is comparatively useless.

(3) A vaccine heated to 60° C., or more, gives rise to a much more severe local and general reaction than one heated to 53° C. for one hour.

(4) The minimum of reaction with the maximum of protection is obtained by the use of a vaccine which has been prepared from a forty-eight hours' growth in broth of an avirulent strain killed by heating to 53° C. for one hour, and 0·3 per cent lysol added to the cool vaccine.

[1] This applies only to a broth vaccine of the *B. typhosus*.

With this vaccine as now in general use such excellent results have been obtained that little difficulty is experienced in getting volunteers. Typhoid inoculation was revived in India in 1904, when Captain Smallman, R.A.M.C., who was attached to the 1st Battalion Royal Fusiliers, arrived in the country with this regiment.

The regiment had prior to embarkation furnished the material for a series of critical experiments to discover the amount of the antitropic substances present in the blood serum after inoculation. Unfortunately, as regards the trial of the vaccine, this regiment was for the first two years in India placed in a station where the men were not exposed to the risks of typhoid infection and, subsequently, when they were moved to a station where enteric was endemic cases occurred as often among the inoculated as among the uninoculated; the fact being that the protection afforded by the original vaccine, then in use, had already vanished. For these reasons the experiment proved abortive and, indeed, in some degree the results retarded the cause of enteric inoculation in India.

A second regiment, the 17th Lancers, was a year or so later sent direct from England to Meerut, a station which, especially from the cavalry lines, had yielded many cases of enteric fever annually. One hundred and twenty-seven men of this regiment had been inoculated twice and twenty-three men once only, while four hundred and forty-three had not been inoculated. The vaccine used was Wright's as modified by the more recent investigations referred to. Sixty-three cases of enteric fever occurred in the regiment within a few months of their arrival; all but two were among the uninoculated. The two cases among the inoculated had only received one dose of vaccine. Not one of those who had been twice inoculated suffered from enteric fever, although all were exposed to the same risks.

This brilliant result, as recorded by Captain Luxmoore, R.A.M.C., the officer on special duty with the regiment, may be said to have re-established anti-typhoid inoculation in the Army, and the measure was thereafter actively pushed. Fortunately, the Commander-in-Chief showed himself to be interested, and a believer in the efficacy of vaccination, and in consequence it was at last well received by the regimental authorities.

At the same time a word of praise must be given to the medical officers who carried out the actual propagandism and inoculation, no light labour, and in the early days (1906) — I speak from personal experience—often a very thankless task.

Some figures are given showing the numbers inoculated during the last five years in India, and are taken from a paper by Colonel Firth published in the JOURNAL OF THE ROYAL ARMY MEDICAL CORPS for June, 1911 :—

	Inoculated	Ratio per 1,000
1906	4,682	66
1910	58,481	823

These figures speak for themselves, and when it is remembered that among the twenty per cent or so not inoculated are included those who have already passed through an attack of enteric fever, and also those men who are beyond the age of maximum susceptibility, we see at once what a remarkable change has been effected. In place of a susceptible community we have an immunized Army. This immunity can be shown directly by the results of the Widal reaction, and also by statistical returns.

AGGLUTINATION TABLES.

73 men tested.

51, i.e. 70 per cent = ± 100.
14 ,, 19 ,, = ± 40.
8 ,, 11 ,, = Nil.

60 men tested.

35, i.e. 58 per cent = ± 100.
19 ,, 32 ,, = ± 40.
6 ,, 10 ,, = *Nil.*

Of these, the majority had been inoculated within twelve months; ten of those who gave a negative reaction had been inoculated more than a year previously.

Of the 335 cases of enteric fever which occurred in 1910, 187 were inoculated men, and 148 were not inoculated. Incidentally, this shows, as everyone is aware, that inoculation is not an absolute protection; but it must be borne in mind that a number of these cases among the inoculated were really paratyphoid fever.

	Admission ratio	Death ratio
Inoculated	3·19	0·37
Uninoculated	12·72	2·06

Of the hundred and eighty-seven men who suffered from enteric fever during 1910 :—

25 per cent had been inoculated one year.
46 ,, ,, ,, two years.
17 ,, ,, ,, three years.
9 ,, ,, ,, four years.
3 ,, ,, ,, more than four years.

Colonel Firth also attributes to inoculation the fact that there is now a definite postponement of the maximum incidence of

enteric fever from the first twelve months of Indian service, as previously, to the second or third year as at present.

The new arrivals are either freshly innoculated or are inoculated soon after landing. The protection lasts two years, but is wearing off gradually during that period. Moral: re-inoculate after thirty months.

TABLE SHOWING THE REDUCTION OF ENTERIC FEVER INCIDENCE SINCE THE INTRODUCTION ON A LARGE SCALE OF ANTI-TYPHOID INOCULATION[1] AND THE ESTABLISHMENT[2] OF THE DEPOTS FOR CONVALESCENTS.

Year	Ratio per 1,000 Admissions	Ratio per 1,000 Deaths
[1] 1906	15·6	3·19
1907	13·1	2·77
[2] 1908	14·6	2·74
1909	8·9	1·58
1910	4·1	0·62
	Actuals.	
1898	2,375	657
1906	1,095	224
1907	910	192
1908	998	191
1909	616	112
1910	335	47

Naini Tal Depot, 1911.

Of 18 enteric fever cases diagnosed by blood cultures	9 were inoculated, i.e. 50 per cent.
Of 39 ,, ,, diagnosed clinically	20 were inoculated, i.e. 50 per cent.
Of 85 paratyphoid fever cases	80 were inoculated, i.e. 94 per cent.

Ten years ago there were more deaths from enteric fever than there were cases in 1910.

A brief account of the history of anti-typhoid inoculation and of its firm establishment has been given, as, although latterly the work of the writer has lain along other lines, yet he is convinced that the reduction of enteric fever in India is in the main due to this practice. And he is also convinced that although inoculation is a "confession of weakness," yet it must always be one of the first weapons to be used when troops are about to be exposed to danger of infection over which we have no control, as when they are engaged on active service in a country where enteric fever is endemic among the inhabitants.

In some quarters, and these high quarters, the reduction of enteric fever incidence in military service has been attributed in

great part, if not entirely, to improved sanitary methods. Undoubtedly there has been of late years a great improvement in the personal hygiene of the individual soldier; this must have had a considerable bearing on the typhoid problem in reducing the opportunities of the carrier. But the writer is convinced that if a carrier was introduced into a station no amount of sanitary effort, as at present understood, would prevent the occurrence of fresh cases of enteric fever, although, of course, cleanliness, public and private, would reduce his opportunities.

It must be remembered that in India at least, apart from the substitution of a "wet method" of conservancy in place of the old dry-earth system, there has been no radical sanitary change of recent years. The latrines and cook-houses and barracks are the same buildings which were in use twenty years ago. It is, I think, a little unfair to our predecessors to say that there has been an enormous improvement in sanitation of recent years in India; and a perusal of an article, which is included among "Echoes from the Past" in the JOURNAL OF THE ROYAL ARMY MEDICAL CORPS of February 1912, will show that officers twenty years ago were just as keen and had also realized the importance of attention to sanitary details.

The difference now is that this keenness and knowledge are not confined to the Medical Corps only, but are shared and appreciated at their true value by the regimental officers who are the executives.

Roberts, in his book on "Enteric Fever in India," points out that sanitary effort has been effective against other "filth" diseases, but that, in spite of all efforts, and they have been very real and energetic since 1890, there has been a gradual and continued rise in the incidence of enteric fever since that time and up to the present (1906), if that incidence is calculated on the numbers of susceptible people exposed.

In his opinion, the only sanitary measure which could have any effect on the incidence of enteric fever would be the introduction of a water-carriage system of sewage disposal, and apart from this "all sanitary effort is mere beating of the air."

In spite of the fact that no water-carriage system of sewage disposal has been introduced, enteric fever has almost disappeared in India in the five years since 1906, when the above opinion was expressed.

It is obvious, then, that we must look beyond ordinary sanitary measures if we wish to discover the reason for this remarkable reduction.

TABLE.—Actual Number of Deaths and Rates per mille, 1862-1900, India.*

Deaths and Death-rates only.

Periods. Average strength	Cholera	Enteric fever	Other fevers	Dysentery, diarrhœa	Total deaths, all causes
1862-70 ..	2,630 ..	1,589 ..	1,441 ..	1,700 ..	13,020
59,827 ..	4·9 ..	2·9 ..	2·6 ..	3·15 ..	24·2
		Total, 10·4 per 1,000.			
1871-80 ..	1,592 ..	1,184 ..	724 ..	946 ..	10,363
56,213 ..	2·8 ..	2·1 ..	1·28 ..	1·7 ..	18·67
		Total, 7·9 per mille.			
1881-90 ..	899 ..	2,342 ..	431 ..	546 ..	8,744
61,399 ..	1·46 ..	3·81 ..	0·7 ..	0·89 ..	14·24
		Total, 6·9 per mille.			
1891-1900 ..	726 ..	4,404 ..	506 ..	638 ..	6,274
68,224 ..	1·06 ..	6·46 ..	0·74 ..	0·94 ..	
		Total, 9·2 per mille.			

* Table taken from "Enteric Fever in India," by E. Roberts.

In 1862-70 the death-rate from cholera was double that from enteric fever, that is, in the pre-sanitary era. In 1891-1900, a period of marked sanitary effort, the death-rate from enteric fever was three times as great as in 1862-70, and six times greater than that for cholera.

To everyone who had studied the question of enteric fever in military service in recent years it was obvious that although improvement in the water supply, etc., had almost eliminated cholera from the list of diseases to which troops were liable, it had had no effect whatever on the incidence of enteric fever. It was obvious, then, that some other cause or causes must be looked for to account for the increasing prevalence of the disease.

The work of Robert Koch and his fellow-scientists in Germany, which has already been referred to, at once directed attention to "man the storehouse of the virus."

The Government of India decided that an inquiry should be instituted on the lines of the German Commission, to determine whether enteric fever was due to the presence of infected persons among the troops. This inquiry was started in 1906, and the writer was fortunate enough to be selected as a member.

Major E. D. W. Greig, I.M.S., who was at the time in England, was directed to proceed to Germany to study the methods employed there at first hand. He made a stay of some months in the country and was given every opportunity to study the work in all its details. On his return to India work on similar lines was at once commenced.

I think there can be no two opinions as to the value of this work, although at the same time I am not prepared, as some officers are, to attribute the reduction of enteric incidence entirely to its effects and that of the depots subsequently established. At the same time no paper dealing with the prevention of enteric fever in military service would be complete which did not deal fully with these researches.

The work was commenced at Meerut in 1906. This station was selected as it had returned a large number of cases of enteric fever for several years. Colonel D. Semple, Director of the Central Research Institute, who shared in all the work of the inquiry, decided to commence by the examination of the excreta of healthy men in units in which enteric fever was endemic, with a view to the discovery of carriers. Out of five hundred men examined one man was found to be excreting the *B. typhosus* in his fæces.

On looking back on this work in the light of recent research, I am of opinion that we were lucky to find even one such case.

It was obvious that this line of work was not likely to prove productive, and as time was limited it was decided to abandon this side of the question and to take up the examination of actual cases of enteric fever, and to follow them up throughout convalescence. This was done in a series of eighty-six cases.

The urine and fæces of these men were examined daily. As three large nine-inch plates of Conradi medium were used for the fæces, and two for each sample of urine, it can be understood that to examine the excreta of a dozen or more convalescents was no light work. Each plate required approximately thirty cubic centimetres of medium, and every likely colony was picked off and investigated.

Of these eighty-six men, ten were found to be passing the bacillus in their fæces or urine for periods longer than six months after defervescence. This gives a percentage of 11·6, a very high percentage indeed; but, as a matter of fact, only two of these men ever became true chronic carriers.

In addition to this work on the fæces and urine which had such a practical bearing on the enteric problem in India, a considerable amount of work was done on the treatment of enteric fever cases by autogenous vaccines (Semple, *Lancet*, 1909). Also in all cases the Widal reactions, opsonic index and bactericidal content of the serum were estimated, with the view of discovering whether such examinations would be of use in the detection of carriers among convalescents and healthy men. The methods of estimating the opsonic index used by the writer in 1907 were as follows:-

Two volumes of serum diluted one-fifth, heated to 60° C. for ten minutes.

Two volumes of washed blood cells.

One volume emulsion of *B. typhosus*.

The whole mixed and incubated for fifteen minutes at 37° C.

An improvised incubator capable of holding eight tubes was utilized and was placed on the work bench.

The sera of a very large number of normal men were examined by this method, and it was found that none gave a higher count than an average of one bacillus per cell. The normal, therefore, was taken as unity, and the index of the patients' serum was recorded as the average number of bacilli ingested per cell. As a rule the patients' serum gave an average of five to ten or fifteen per cell, according to the stage of their illness, the highest point being reached about the third week of the fever. In one case which on admission gave a count of about ten bacilli per cell, the index dropped gradually from day to day and was entirely negative on the day of death, which took place from toxæmia about a week after admission to hospital.

An alternate method used by the writer at this time (1907) was to treat opsonic estimations on the same principles as agglutination tests, by means of dilutions; the highest dilution which reduced immune serum to the level of the normal being taken as the end point. This method was abandoned, as it was too laborious and occupied too much time.

The results so far as the detection of carriers was concerned were not satisfactory, as many convalescents who were not carriers showed raised indices and several carriers gave less than the normal counts.

One curious phenomenon noted was that the serum of one worker, who was being used as a "normal man," quite suddenly began to give a raised index to typhoid, and continued to rise till the count was equal to that of a severe case of enteric fever in the third week, i.e., an average of about twenty bacilli per cell. The blood and excreta of this man were carefully examined, but with negative results, and he was apparently in his usual good health.

Later on the writer was using his own serum as normal when suddenly the index commenced to rise and rose gradually for about a week, and then gradually subsided again to the normal. It would be well to keep such contingencies in mind, as otherwise they may give rise to error.

The serum of another worker, on the contrary, remained at unity for months without any variation.

The bactericidal content of the serum was also estimated by the usual method of utilizing a diluted broth culture of typhoid against varying dilutions of the sera. This method occasionally gave concordant results, but sometimes the normal gave a higher reading than the immune serum, this being due to a lack of free complement in the latter. This difficulty was overcome by heating both sera and adding equal quantities of fresh guinea-pig serum to the normal and to the immune.

The result of the work at the Central Research Institute and elsewhere was to direct attention to the fact that young soldiers, after an attack of enteric fever, may continue to excrete the bacillus in their fæces or urine for months, and in rare cases for years. When this fact was realized it was at once evident that the system in vogue at the time, of returning men to duty as soon as they were physically fit, was one attended with a considerable degree of risk. At least ten per cent of such men would be infective for varying periods.

It was therefore decided, as it would not be feasible to keep all men in hospital until such time as they might be considered non-infective, to establish depots in the Hills, to which all men convalescent from enteric fever should be sent, and where they would remain as long as they were found to be infective, and for at least three months if all examinations were negative.

It was thus proposed to meet two requirements :—

(1) To secure a place where convalescents from enteric fever would have an opportunity under favourable climatic conditions of recruiting their strength, and where they might gradually return to full duty by a series of carefully graduated exercises and drills.

(2) To establish a central laboratory where the excreta of these men could be bacteriologically examined, with a view to the detection of carriers and the quick return of the non-infective to duty consistent with their physical fitness. At the same time to continue the work begun at Kasauli on the duration of infectivity, and the identification of bacilli, etc.

The place selected for the first depot was at Naini Tal, in the Kumaon Hills. These barracks had been used for many years in the summer time for the reception of sickly men from the 7th and 8th Divisions. Convalescents from enteric fever had been accommodated in one block, and convalescents from malaria, etc., in the others, while healthy men on furlough were placed in tents.

It was now proposed to reserve the entire barrack-room accommodation (124) for enteric fever convalescents alone, from the 1st,

2nd, 3rd, 7th, 8th, and part of the 5th Divisions—that is to say from practically the whole of Northern India, from Nowshera in the north to Calcutta in the east, and as far south as Jubbulpore. Latterly men were also received from the 4th Quetta Division.

Unfortunately, when selecting these barracks for this purpose the local civil authorities were not consulted—Naini Tal is the summer headquarters of the United Provinces Government—with the result that there was continual friction and misunderstanding with the local authorities. One very highly placed civil medical man was of the opinion that *all* the men sent up to the depot were "carriers" and that all were excreting "millions of bacilli," and advised the Governor, Sir John Hewitt, in these terms.

An outbreak of enteric fever in the civil community was attributed to the depot, although none of the men were permitted to enter the town, which was distant one mile and a half from the depot and some hundred feet above it.

The depot was supplied by the same piped water supply as the town, and one medical man gravely considered the possibility of typhoid germs being placed in the water at the depot end of the pipe and travelling up against a strong head of water to the source of the distribution. One lady, the wife of a Surgeon-General, A.M.S., now retired, was heard to say that her "dear Naini would be quite spoiled," as she could no longer take a favourite walk for "fear of meeting one of these dreadful carriers."

It will be understood that the work of the depot, at no time of the most congenial, was not rendered any the more pleasant by reason of the local prejudice, which would never have arisen had the purpose and scope of the work been explained before its inception. But it is pleasant to be able to relate that the depot survived several "inquiries," and in 1910 it was visited by the Sanitary Commissioner with the Government of India, the late Lieutenant-Colonel Leslie, I.M.S., who came in the first instance at the request of the local government to condemn, but remained to praise, in that he not only exonerated the depot from all blame, but asked that he might be supplied with a detailed account of the plan of work, with a view to the institution of similar depots on the civil side.

No structural alterations were made in the barracks or latrines, but a boiler for excreta was provided and a steam sterilizer (Thresh-Delépine) was also sent up.

Saponified cresol was used in the latrines and, after boiling, the excreta were trenched on the hillside about half a mile below the barracks.

The men began to arrive at the depot on April 8, 1908, and have been passing up and down in constant but ever lessening numbers up to the present.

There was fortunately a very good and fairly well equipped laboratory situated near the Station Hospital, and about a mile from the depot. This laboratory was used in the hot weather by the Sanitary Officer of the 8th Lucknow Division, Major J. C. Morgan, R.A.M.C. No grant was given for the laboratory work of the depot, but it was stated in the preamble that material would be supplied by the Sanitary Officer, 8th Division, from his grant of thirty rupees *per mensem*. It may be said here that the bacteriological work could neither have been commenced nor carried on had it not been for the very cordial co-operation of the Sanitary Officer and his successor, Major J. C. Weir, R.A.M.C., who not only gave up practically the whole of their grant for this work, but also expended a considerable amount in excess, which they, only after a great deal of trouble, were able to recover from the financial authorities.

Lieutenant-Colonel D. Semple, Director of the Research Institute, Kasauli, who was interested in the carrying on of the work, contributed from time to time grants of material, without which it would not have been possible to do work on any scale at all, and indeed even with this assistance the work, as will be seen later, was sadly in arrear by the end of 1908.

With regard to the administration of the depot, it must be noted that the men were discharged from hospital before they left their stations and were provided with uniforms by their own units. They did not rejoin their regiments, but proceeded direct from hospital to the train and came straight up to the depot without passing through the civil station of Naini Tal. The original rule was that no man should be discharged from hospital until he had been free from fever for six weeks, and only if he was considered fit to travel and to eat ordinary rations on arrival at the depot. In some instances men who had never been on solid food and had only just got out of bed for the first time were sent up to the depot, although in many cases the railway journey occupied two or three days, followed by a *tonga*, or pony ride, of fourteen miles.

If possible, several men were sent from one station at the same time, and they were invariably accompanied by a non-commissioned officer.

Strict rules were laid down as to the necessity of care in regard to the disposal of the excreta of these men and all had received a course of urotropine.

The depot was run on a military footing, the staff consisting of a commandant, a medical officer, an adjutant, one subaltern (company officer), a quartermaster-sergeant, and an assistant surgeon. Discipline in the depot was carried out by the non-commissioned officers among the convalescents. There were as a rule a good supply of sergeants, as they have a way of avoiding inoculation and acquiring enteric fever. At one time, out of one hundred and twenty men in the depot, twenty-five were non-commissioned officers. All duties in the depot, such as guards, etc., were carried out by the convalescents themselves, and including the assistant surgeon who was quartered in the depot every man there was an enteric convalescent.

Latterly (1910) owing to lack of convalescents some difficulty was experienced in carrying on the duties.

The men usually arrived at the depot in the afternoon and were seen the following morning by the medical officer, and as a rule were excused duty for ten days or, if considered fit, might be allowed to do light duty. In some cases it was found necessary to admit the men to hospital for a few days. At the same time a capsule of blood and a blood film were taken from each man and the agglutination reaction estimated. For a time also a sample of urine from each man was examined, but owing to lack of proper equipment and the increase in the numbers of men this had to be given up. But latterly (1910) it has been possible to examine the urine and fæces of each man for a week after arrival.

The fresh arrivals were seen daily for ten days by the medical officer, and if considered fit were at the end of that period put on to light duty for another ten days, and then were allowed to do full duty.

The duties at the depot consisted of a carefully graduated scale of drills, including light marches, short parades, route marches, musketry, signalling, physical exercises, etc. Hockey and foot-ball were also allowed for those who were considered fit.

The result of this system was that at the end of a stay of three months or so, the men as a rule were fit and well and ready to take their places again in the ranks of their regiments, instead of, as in the case of men returning direct from hospital, requiring to be retaught their drills. The rapid improvement in physique of nearly all of the men was most gratifying, and apart from carriers no man was invalided home on account of ill-health, and only one death occurred in three years.

Several men with "enteric leg" were returned to their stations

after being in the depot for some months (twelve in one case) *in statu quo*, although they were able to carry out light duties all the time—one was a most efficient billiard-marker.

The method of collecting samples of urine and fæces for examination was as follows : ten, fifteen, or twenty men, as the case might be, were told off by the orderly serjeant to attend at the inspection room at 5 p.m. and again at 7 a.m. the next morning. These attendances were looked upon as parades and non-attendance as a crime and punished as such by the officer commanding depot. At 5 p.m. each man was given a small lidded tin, provided with a spoon, and was instructed to collect and bring to the hospital on the following morning a sample of his fæces. These tins were collected by the orderly on duty and packed for conveyance to the laboratory. At the same time (morning) each man passed a sample of his urine into a sterile bottle, which was also labelled and packed and carried at once to the laboratory. Here sufficient plates had already been poured to deal with the number of samples expected.

Conradi's original blue medium was used, and about two litres were used daily. A small portion (about one gramme) of the fæces was emulsified in five cubic centimetres of tap-water, and the mixture allowed to stand for about one hour; then two or three drops of the supernatant fluid were placed on a plate and spread with a glass spreader, which was again used to inoculate a second plate. The remainder of the emulsion with the glass tubes and rods and tins were placed in a basin of cyllin and the whole boiled for ten minutes. The urine was plated by placing one or two drops on a plate and allowing them to run over the surface. It was found that by getting the men to pass the urine directly into the sterile bottles and plating within a couple of hours or so, sterile plates were invariably obtained unless the man happened to be a carrier.

The same batch of men paraded for five days in one week, and again for five days in the next week, ten examinations in all. If the results of these examinations were negative the man was allowed to return to his station, provided he was otherwise fit and had been more than three months in the depot.

It is not contended that every carrier could be detected by this method, but as all the men before examination had been four or five months free from fever, it was regarded as the best that could be done under the circumstances. Later on, with more adequate equipment, it was possible to examine each man for a week after arrival and again for a fortnight before his return to his station.

Two known intermittent fæcal carriers were examined by the

two weeks method, and both were detected as passing *B. typhosus*, one twice during the ten examinations and one once.

Men who ultimately become chronic carriers are almost invariably *constant* carriers in the early months, although they may become intermittent later on. Thus the weekly examination on arrival was a great help in indicating the men who ought to be followed up carefully. At the same time, it must be admitted that in one instance a man was examined for a week on arrival and all examinations were negative, yet *B. paratyphosus* A was recovered from his fæces on several occasions two months later.

The majority of the men did not like to be kept in the depot, and it was a common thing to have men attending the sick parade in the morning, and on being asked what was the matter, saying, "Beg pardon, sir, might I be put on tubes?" meaning, could they be examined so that they might return to their stations. All the men were under the impression that they were infected—"germy" as they called it—on arrival and that it was not till they had been examined for some time that the germs disappeared. This idea was encouraged, as it ensured that each man was careful to bring a sample of his own fæces.

Known carriers were required to pass their excreta in hospital, and any suspected carrier had a sample taken from his excreta by the orderly in addition to the sample supplied by himself.

During the three years, 1908-11, no case of deliberate substitution was encountered.

The method of examination of the plates prepared from fæces or urine was to pick off all non-lactose-fermenting colonies, i.e., all blue colonies. Many of these proved on further investigation to be capable of splitting lactose. Small drops of high titre serum diluted 1 in 60 were placed on a glass slide, and a small portion of the colony to be tested was rubbed up in the drop.

The sera used were for typhoid, paratyphoid A and paratyphoid B. Great difficulty was experienced in obtaining serum for A, as the Berne Institute does not stock it; but some was obtained from Kasauli, and an animal (rabbit) was immunized at Naini Tal, although several were killed in the process. Apparently, susceptibility is increased rather than diminished by repeated doses of this bacillus, and any attempt at increasing the dose may be followed by a complete disappearance of antitropic bodies, and possibly the death of the animal.

Whether the colony was agglutinated (macroscopically) or not by the typhoid serum, it was invariably also tested in A and B

serum, and a colony which was immediately agglutinated by one serum and made an even grey emulsion in the other two, was always regarded hopefully.

A curious fact is that the majority of freshly isolated paratyphoid A bacilli are agglutinated in the typhoid serum as well as by their homologous serum, although the converse is not the case. If the colony was found to agglutinate in any one serum, in two, or in all three, it was planted out on an agar slope for further investigation. On the following day it was retested from the agar culture in the sera, and a series of "sugar" tubes was inoculated.

The following were used as routine measure:—

Maltose.

Lactose.

Glucose.

Mannite.

Peptone water.

Milk (litmus).

Cane-sugar.

Neutral red agar (glucose), shake culture.

These were examined daily for a week and the results recorded from day to day. If a tube was found to be fermented a plate culture was made to show whether this was due to a possible contamination.

A second agar culture was made and agglutination tests were carried out from this.

(1) With the serum of the man from whose excreta the bacillus had been isolated.

(2) With the serum of a carrier or of a man whose agglutination titre for typhoid was known.

(3) With the specific sera.

All these estimations were made in sedimentation tubes with emulsions made in saline solution and standardized by counting or opacity.

Absorption tests were used in all cases where there was any doubt, and latterly in every case.

These latter tests have shown that there are bacilli to be met with in human fæces which will apparently give all the reactions of a true typhoid bacillus and are agglutinated by human serum, and yet "are not what they seem." At the same time, the writer has met with bacilli which obviously did not belong to the typhoid group (fermentation of cane-sugar) and yet absorption with a strong emulsion of these bacilli removed all the agglutinins from the specific serum (paratyphoid B).

I do not think, indeed I may say after an experience of some years, I know, that it is not sufficiently realized how closely the bacilli of the typhoid group may be simulated by other bacilli in human excreta. These bacilli are not commonly met with, any more than bacilli of the typhoid group are commonly met with, but they do occur every now and then and give rise to mistakes.

Quite a number of men were sent up to the depot as "carriers," yet in no single instance did these men prove to be true carriers, although in the excreta of several of them bacilli closely simulating typhoid or paratyphoid bacilli were met with. As a rule, these bacilli were agglutinated by one or other of the specific sera, but differed from the typhoid group in their sugar reactions. In one instance a man was invalided home by the writer as a carrier, although on looking back I am quite convinced that he was never a true carrier, but was excreting one of these readily agglutinable bacilli resembling typhoid. Mention is made of this case under "Notes on Carriers."

Some examples of bacilli which simulated bacilli of the typhoid group are given below.

It is somewhat difficult to describe on paper the reactions of these various strains of bacilli, as some apparently gave the true sugar reactions of the typhoid group, and were also agglutinated by one or other of the specific sera, and could only be condemned from the quantitative point of view, as it were. Others, when the results are put on paper, are apparently far removed from the typhoid group, and yet it was only after prolonged work that these could be excluded.

Reactions of bacilli isolated on the same day from the fæces of Private H. and Private N.

Clear blue colonies; clump at once in A serum, not in typhoid.

Sugar Reactions.

Peptone Water Tubes.

	24 hours	48 hours	One week
Lactose	*Nil*	*Nil*	*Nil.*
Glucose	,,	Acid	Acid.
Mannite	,,	,,	,,
Cane-sugar	,,	*Nil*	*Nil.*
Milk	,,	,,	Acid.
Peptone	,,	,,	No indol.

Agglutination Reactions.

Serum of Private H., an enteric convalescent.

	Dilutions				
	10	20	40	100	200
H.'s bacillus	+	+	+	+	+
N.'s bacillus	+	+	+	+	+
Paratyphoid A	–	–	..	..	..

Private N.'s serum. Enteric convalescent.

	Dilutions			
	10	20	40	100
H.'s bacillus	+	+	+	+
N.'s bacillus	+	+	+	+
Paratyphoid A	−	−	..	..

Private B.'s serum. Paratyphoid convalescent.

	Dilutions			
	10	20	40	100
N.'s bacillus	+	+	±	±
H.'s bacillus	±	+	±	+
Paratyphoid A	+	±		−
Typhoid	+	+	±	±

Private B.'s serum after absorption with N.'s bacillus.

	Dilutions			
	10	20	40	100
N.'s bacillus	−		−	
H.'s bacillus	+	+	+	+
Paratyphoid A	±	±	−	..
Typhoid	+	+	±	±

Private B.'s serum after absorption with H.'s bacillus.

	Dilutions			
	10	20	40	100
N.'s bacillus	+	±	±	±
H.'s bacillus	−	−	−	−
Paratyphoid A	+	±	−	..
Typhoid	±	+	±	±

These were interesting bacilli, as it was found that they were immediately agglutinated by every sample of human serum they were tested against and yet were not agglutinated in a control tube of saline solution. Absorption with one bacillus only removed the homologous agglutinin, showing that this was specific, and although both bacilli were agglutinated by paratyphoid A serum they could not remove the agglutinins for this bacillus from the serum of a convalescent. Had it not been possible to use specific sera of high titre these bacilli would have resembled typhoid bacilli very closely as they were agglutinated by the sera of convalescents.

Colony isolated from the fæces of Private W.

Agglutinated at once macroscopically by A serum, not by typhoid or B.

Sugar Reactions.

	24 hours	Ten days
Lactose	*Nil*	*Nil.*
Mannite	A + G ¾*	A + G.
Glucose	A + G. Total*	A + G.
Cane-sugar	*Nil*	*Nil.*
Milk	Acid	Acid.
Peptone	.. .. ..	No indol.

* Too much gas for paratyphoid A.

Did not remove the agglutinins from paratyphoid A serum by absorption.

Colony isolated from the fæces of Private G.

Agglutinated at once in A serum, not in typhoid or B.

Sugar Reactions.

Lactose	*Nil.*
Glucose	A + G ¾.
Mannite	A + G. Total.
Cane-sugar	*Nil.*
Milk	Acid.
Peptone	*Nil.*

The colonies of this bacillus on a Conradi plate, where they had room to grow out, were too large and coarse for the typhoid group, but the smaller colonies were apparently quite typical.

Absorption Tests.

Untreated high titre serum specific for A bacillus.

Stock paratyphoid A	Immediate clumping.
K.'s bacillus	,, ,,
W.'s bacillus	,, ,,
G.'s bacillus	,, ,,

Serum A after absorption with W.'s bacillus.

Stock paratyphoid A	Immediate clumping.
K.'s bacillus	,, ,,
W.'s bacillus	No clumping.
G.'s bacillus	Immediate clumping.

Serum A after absorption with G.'s bacillus.

Stock paratyphoid A	Immediate clumping.
K.'s bacillus	,, ,,
W.'s bacillus	,, ,,
G.'s bacillus	No clumping.

Specific A serum after absorption with K.'s bacillus.

Stock A	No clumping.
K.'s bacillus	,, ,,
W.'s bacillus	,, ,,
G.'s bacillus	,, ,,

This series of experiments showed that K.'s bacillus was a true paratyphoid bacillus, as it removed both the specific and group agglutinins from the serum, and also showed that W.'s and G.'s bacilli were not paratyphoid bacilli, as absorption with these bacilli removed only their particular group agglutinin and did not touch the specific.

Another interesting bacillus isolated from fæces gave the following reactions:—

	Peptone water		Nutrose water	
	24 hours	48 hours	24 hours	One week
Lactose ..	*Nil* ..	A + G ..	*Nil* ..	*Nil.*
Glucose ..	A + G ..	A + G ..	A + G ..	A + G.
Mannite ..	A + G ..	A + G ..	A + G ..	A + G.
Cane-sugar ..	A + G ..	A + G ..	*Nil* ..	*Nil.*
Milk ..	Acid ..	Acid ..	Acid ..	Acid.

It will be noted that but for the milk tube this bacillus, in the sugars prepared with nutrose water, gave the reactions of paratyphoid B, but in the peptone water sugars gave the reactions of the *colon* group. This bacillus was agglutinated at once in normal saline solution and consequently by all sera diluted with normal saline. Absorption with this strain of bacillus of sera specific for typhoid, paratyphoid A and paratyphoid B removed all the agglutinins from the last but did not touch the other two.

Examples might be multiplied indefinitely, as it was rare for a day to pass without encountering one of these "simulating" bacilli; but enough has been said to show how closely some bacilli may resemble the typhoid group and also how common these bacilli are in the excreta of enteric convalescents.

It might be urged that such careful investigation of each bacillus is not necessary, but it is undoubted that without some such system mistakes will occur. Fortunately, these mistakes when they are made are, as a rule, on the safe side, i.e., bacilli resembling typhoid are taken for typhoid; the converse is not so likely to occur.

The method used in the absorption tests was as follows:—

About 0·5 c.c. of the serum was placed in a watch-glass and the whole of an agar culture of the bacillus to be tested was rubbed up in the serum to form a very thick emulsion. This was then pipetted into a narrow tube which was plugged with cotton-wool, placed in the incubator at 37° C. for three hours, then removed and centrifuged and the clear supernatant fluid used for the subsequent tests, either in sedimentation tubes or simply as a drop on the slide for the macroscopic method, a portion of the cultures being rubbed

up in the drop. By this method quite a number of bacilli could be tested at one time against the specific sera.

There is little doubt that these "typhoid simulators" account for many erratic findings of typhoid or paratyphoid bacilli in the fæces of healthy men, in flies, etc., and work on the detection of carriers should only be attempted by those who have had special experience.

During the nine months of 1908, 310 men arrived at the depot. With the equipment available it was only possible to examine the excreta of 10 men daily, and as it was considered necessary to examine each man at least ten times, it will be understood that it was not possible to keep abreast of the work. Only 190 were examined and returned to their stations, leaving 120 men in the depot at the end of the year still to be examined and about 80 men waiting to be sent up from the plains, for whom there was no accommodation. As soon as the new equipment, consisting of one gross smaller plates (six inch) and a larger incubator, capable of holding eighty such plates, arrived, the number of examinations was doubled and the arrears were soon wiped off.

But it was not until the end of 1909 that it was possible to examine men at once on arrival and for a fortnight before their return to the plains.

During the summer months the men who had been longest in the depot were accommodated in tents, thus making room for fresh arrivals from the plains. At one time there were close on two hundred convalescents in the depot. As already pointed out, had it not been for the reduction in enteric incidence brought about by inoculation, it would have been quite impossible to deal with the convalescents from so many stations. Indeed, in former years, one large station, such as Lucknow or Umballa, would have required a depot of its own, with a whole-time medical officer, whereas the present depot was run by a medical officer in addition to his ordinary duties, which duties in previous years prior to the establishment of the depot had given employment to two medical officers.

In 1909 the medical officer in charge of the depot was struck off duty at the Station Hospital, and in 1910 a second medical officer was appointed for duty in the laboratory and depot.

Out of the 190 men whose examinations were completed by the end of 1908, 5 carriers were detected. Notes on the cases of these men are appended. Two of these men came from Kasauli and were already known to be carriers, 1 was returned in error, 2 only were definite chronic carriers from among

the new arrivals, thus giving a percentage of 1·05. This I consider about the true percentage of definite chronic carriers among young men convalescent from enteric fever. One thousand four hundred and seventy-two samples of fæces were examined and one thousand four hundred and forty-eight samples of urine. Not reckoning repeated examinations of the same man, the typhoid bacillus was only recovered once out of all these samples of urine. One of these men was a fæcal carrier, the other urinary. During the three years following up to the end of the year 1911 no other chronic urinary carrier has been detected either at the Naini Tal or the Wellington depot. It may be urged that some may have been overlooked, but with the urine of this chronic carrier it was only necessary to place a single drop on a plate to ensure a pure culture of the *B. typhosus* on every occasion.

Again, when examining the urine of some boys who were suffering from enteric fever, the *B. typhosus* was recovered from the urine in fifty per cent of the cases during the fever and in early convalescence, although all ceased to pass the bacillus within a month of the cessation of the fever.

Apparently among young men the "chronic urine carrier" is a very rare accident indeed, and fortunately so, because he must necessarily be a greater danger to his comrades than the fæcal carrier.

As at this time (1908) there were two men in the depot, the one Br. S., a chronic carrier (urine) of *B. typhosus*, and the other Gr. C., a chronic carrier (fæcal) of the same bacillus, it was decided to carry out some experiments, with a view to determining the viability of this bacillus under as nearly as possible natural conditions. The results of these experiments have already been published, but are briefly recorded here, as they have a very direct bearing on the problem of the causation and prevention of enteric fever in military service.

Before recording the results of these experiments, a word of explanation is necessary as to the expression used "*B. typhosus* in pure culture." This expression, when referring to a plate culture from fæces is only a relative term and depends on the medium employed.

For instance, if an equal amount of a fæcal emulsion from a typhoid carrier is plated out on :—

(1) Ordinary agar,

(2) McConkey's bile salt neutral red agar,

(3) Conradi's crystal violet medium,

the results might be expressed as follows :—

(1) Plate almost overgrown principally by *coli* colonies; numerous minute typhoid colonies.

(2) Not so many *coli* colonies; typhoid colonies numerous and larger.

(3) Practically a pure culture of typhoid: a few scattered *coli* colonies.

This expression, then, refers to the appearance of a sample of fæces (Gr. C.'s) plated on Conradi's blue medium, and does not imply that there were no *colon* bacilli in the sample of fæces.

Experiment I.

To determine the viability of the *B. typhosus* when voided directly on to the ground in the urine of a chronic carrier.

Br. S. micturated on to a patch of dry ground in the depot.

Result.—Six hours after pollution the *B. typhosus* was easily recovered from the washings of ½ grm. of soil, but thirty hours later could not be found. A not unimportant detail is that the soil was still damp with urine for some hours after the *B. typhosus* had disappeared. The methods used were rough, and intentionally so, as it was not proposed to discover how long a single bacillus could be found, but simply to show how long the urine of a chronic carrier is dangerous after it has been voided on the ground. For this reason more than 0·5 grm. of soil was not used for the experiment. As it was considered that possibly the sun and light had determined the rapid disappearance of the bacillus, the experiment was repeated, but on this occasion Br. S. passed his urine on the ground in the corner of a dark hut. But here also the bacillus could only be recovered from the washings of a small quantity of soil up to twenty-four hours after pollution and not later.

Practically the same result was got with polluted soil kept in the laboratory at room temperature exposed to light but not to sun. The *B. typhosus* was readily found for a few hours but not after one day.

These experiments show that the surface soil is dangerous for some hours, but it is no longer so when dry enough to be blown about as dust.

Very different results were obtained when towelling was soiled with the urine and allowed to dry, as in this case bacilli could be recovered up to ten days or more after the soiling of the material. The reason for this is that on the towels there can be no multi-

plication of *coli* or soil bacteria, and hence no rapid destruction of pathogenic bacilli.

Thus it is evident that the few drops of urine on the underwear of the carrier are a source of greater danger than the bulk voided on to soil or in a latrine pan or urinal tub.

It is a well-known fact that soldiers freely lend and freely take the loan of one another's clothing, *i.e.*, football shorts, etc., and thus many obscure cases of enteric fever may be accounted for.

The experiments with the fæces were also devised to answer questions which are frequently propounded.

If a carrier passes a stool into a dry-earth receptacle, how long does the stool remain infective?

The answer is contained in the following table :—

TABLE.

Date		Result
12.11.8	Sample plated out	B.T. numerous.
13.11.8	,, ,,	B.T. + 8 colonies second plate.
15.11.8	,, ,,	B.T. *Coli* increasing.
16.11.8	,, ,,	B.T. + ,, ,,
17.11.8	,, ,,	B.T. + ,, ,,
18.11.8	,, ,,	B.T. − from outer crust.
19.11.8	,, ,,	B.T. + from centre.
22.11.8	,, ,,	B.T. + ,, ,,
25.11.8	,, ,,	B.T. + ,, ,,
27.11.8	,, ,,	B.T. −
29.11.8	,, ,,	B.T. −
1.12.8	,, ,,	B.T. −
		Not later.

The *B. typhosus* was recovered in large numbers for the first two or three days and it could be recovered from the outer portions of the fæcal mass up to the fifth day, but not later. From the centre of the mass it could be recovered for a fortnight, but not later, even when large pieces of the fæces were emulsified and several plates prepared from the emulsion.

It was found that when *small* portions of the fæces were smeared on blanket, the pathogenic bacteria rapidly disappeared (three to four days), provided that the weather was warm and permitted of the multiplication of *coli*. The fæcal experiments differ from the urine in this point, namely, that in the former, *coli* invariably accompanies the *B. typhosus*.

These experiments with blankets and others on the same lines done at Kasauli and Poona throw a very lurid light on the famous South African blankets, from which "millions" of typhoid bacilli were stated to have been isolated many months after a very problematical pollution.

When the weather is too cold to permit of multiplication of *coli* quite other results are obtained, as in one instance typhoid bacilli could be recovered from contaminated blanket one month after the experiment was commenced.

Two years after the above experiments were performed it happened that there were in the depot at the same time two men, both chronic carriers (fæcal), the one of the *B. typhosus* and the other of the *B. paratyphosus* A. It was thought that it would be useful to repeat some of the previous experiments, using the fæces of the two men as material. The purpose of these experiments was two-fold :—

(1) To ascertain whether, as is generally supposed, the *B. paratyphosus* is more resistant in nature outside the human body than the *B. typhosus*.

(2) To ascertain whether with the added experience of two years' work, and with the aid of the newer media (Conradi's green medium and Fawcus and Padlewski's modifications of the same), it would be possible to recover the bacilli more readily and over a longer period.

It may be said at once that both these questions had to be answered in the negative.

Experiment I.

Private H. : Typhoid carrier ; entire stool passed into gumlah filled with sawdust.

Private M. : Paratyphoid carrier ; entire stool passed into gumlah filled with sawdust.

From time to time 0·5 grm. of fæces was removed, emulsified in tap-water and plated out.

Table I.

	H.'s fæces ; typhoid	M.'s fæces ; A
5.9.10 ..	B.T. numerous..	Paratyphoid A numerous.
7.9.10 ..	A few B.T. ; great increase of *coli*	A few paratyphoid A ; many *coli*.
12.9.10 ..	No B.T. ; pure *coli*	No paratyphoid A ; pure *coli*.
14.9.10 ..	,, ,, ...	,, ,,
21.9.10 ..	,, ,, ...	,, ,,

After one week neither *B. typhosus* nor *B. paratyphosus* A could be recovered from a gramme of fæces taken from the centre of the mass.

Experiment II.

H.'s total stool placed in a sterile stoppered bottle.

M.'s total stool placed in a sterile stoppered bottle.

TABLE II.

	H.'s fæces; B.T.	M.'s fæces; paratyphoid A
20.9.10 ..	225 colonies; no *coli* ..	226 colonies; numerous *coli* colonies.
21.9.10 ..	Nearly pure culture B.T. ..	Failed to find paratyphoid A; numerous *coli*.
23.9.10 ..	32 colonies B.T.	No paratyphoid A; pure *coli*.
26.9.10 ..	A few B.T.	,, ,,
27.9.10 ..	No B.T...	,, ,,
28.9.10 ..	A few B.T.	,, ,,
30.9.10 ..	,, ...	,, ,,
1.10.10 ..	No B.T.; pure *coli* ..	,, ,,
3.10.10 ..	,, ,, ..	,, ,,
7.10.10 ..	,, ,, ..	,, ,,

Ten days after the fæces had been placed in the bottle the *B. typhosus* could be recovered, but not later, whereas the *B. paratyphosus* A could not be found after the third day.

The only difference in the conditions was that at the commencement of the experiment *coli* colonies were much more numerous in the fæces of the paratyphoid carrier than in that of the typhoid carrier.

By a curious coincidence typhoid bacilli and paratyphoid were present in almost identical numbers. During the time that this experiment was being carried out the weather was warm and moist and *coli* multiplied rapidly at room temperature. It is in my opinion this factor which determines the length of life of the *B. typhosus* in nature.

EXPERIMENT III.

During December, 1910, when the temperature rarely rose above 45° F., and multiplication of *coli* did not take place, some further experiments were undertaken with the fæces of Private B., East Surrey Regiment, a chronic carrier of the *B. typhosus*.

A portion of the fæces of Private B. was placed in a stoppered bottle in the godown; 0·5 grm. was removed and emulsified and plated out from time to time.

Results.

23.12.10	3,000 typhoid colonies; four *coli* colonies, "pink" variety.
26.12.10	Typhoid in pure culture.
27.12.10	,, ,,
28.12.10	,, ,,
29.12.10	,, ,,
2.1.11	,, ,,
3.1.11	,, ,,
5.1.11	About twelve *coli* colonies; rest of plate typhoid.
9.1.11	More *coli* colonies.
11.1.11	*Coli* more numerous; great reduction of typhoid colonies; weather warmer.
12.1.11	Typhoid numerous.
13.1.11	,, ,,
	Typhoid still present in March, 1911.

This experiment shows that when the weather is too cold to allow of multiplication of *coli*, *B. typhosus* can be found in the fæces for many days, up to eighty days in the above experiment.

Experiment IV.

This experiment was undertaken to compare the above results with those obtained when using laboratory cultures in artificial media.

Two loopfuls of twenty-four hours' broth cultures of *B. typhosus*, *B. paratyphosus* A and *B. coli* were placed as follows :—

In 10 *c.c. of peptone water.*

Tube 1, two loopfuls of B.T. + two loopfuls *B. coli*.
Tube 2, two loopfuls of B.T. + two loopfuls *B. paratyphosus* A.
Tube 3, two loopfuls of *B. paratyphosus* A + two loopfuls *B. coli*.

These peptone tubes were not incubated, but were placed in racks on the bench exposed to bright daylight but not to sun.

Results.

10.10.10. One loopful from each tube plated out.
Tube 1, B.T. present ; *B. coli* predominates.
Tube 2, B.T. and *paratyphosus* A both present.
Tube 3, *paratyphosus* A present, but *B. coli* predominates.
3.1.11.
Tube 1, pure culture *coli* ; no *B. typhosus*.
Tube 2, B.T. and *paratyphosus* A both present.
Tube 3, pure culture *coli* ; no *paratyphosus* A.

The colonies of typhoid and paratyphoid could be quite easily distinguished from one another ; the typhoid colonies were larger, whiter, and more opaque than the paratyphoid, which were small, blue, and transparent. As a general rule it is quite impossible to tell colonies of typhoid from paratyphoid. Typhoid and paratyphoid were present in equal numbers.

The above results have been confirmed by no less an authority than Colonel Horrocks, who, in a paper on the viability of the *B. typhosus*, Journal of the Royal Army Medical Corps, March, 1911, refers to our work, and in the conclusion of his article says: "These experiments (*i.e.*, his own) seem to indicate that the duration of life of the *B. typhosus*, as at present recognized, is very short under natural conditions," and again, "If the dejecta contain many *B. coli*, millions of typhoid bacilli disappear in two or three days," a conclusion which, as will be seen above, I had already arrived at and had embodied in my Annual Report for 1910, submitted in January, 1911.

(*To be continued.*)

Clinical and other Notes.

ON THE *DIPLOBACILLUS LIQUEFACIENS* OF PETIT: WITH REPORT OF A CASE SHOWING UNUSUAL DISTRIBUTION OF THIS ORGANISM.

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It is passing strange that the discovery of the causal organism of diplobacillary conjunctivitis was delayed until the year 1896 to be independently discovered in that year by two separate observers, namely, Morax in Paris and Axenfeld in Marburg, especially when we consider that diplobacillary conjunctivitis constitutes one of the most commonly occurring infections of the eye, with a world-wide geographical distribution; that the organism is a large and distinctive diplobacillus readily stained by simple dyes like methylene blue and present in abundance in the conjunctival discharge of affected cases, being easily seen in smear preparations thereof; and, also, that it can be cultivated without difficulty on blood serum, in which it liquefies in such a way as to bring about the characteristic pitting in this medium.

A very closely allied organism which may be considered as a variant of the diplobacillus of Morax-Axenfeld was discovered in 1898 by Petit in three cases of painless superficial serpiginous hypopyon-keratitis.

Morphologically and tinctorially the diplobacillus of Petit is indistinguishable from that of Morax-Axenfeld, but is distinguished from it by its greater range of growth both with regard to media and temperature, growing readily on the common media such as agar and gelatine, the latter of which it liquefies, and at temperatures ranging from room temperature to blood-heat. Hence in practice it suffices when a blood-serum liquefying diplobacillus is met with to inoculate an agar tube, when growth indicates the Petit type, and its absence the Morax-Axenfeld type. The frequency of diplobacillary infection of the conjunctiva may be gathered from the fact that Pflüger and Simon found the diplobacillus in about ten per cent of all patients in Berne, whilst Erdmann encountered 342 cases in Rostock in five years, Axenfeld 529 cases in Freiburg in four years, and over 500 cases were observed in the University Eye Clinic in Bonn in the course of a single year.

During the last two years I have cultivated the diplobacillus thirty-eight times at the Royal Westminster Ophthalmic Hospital, being in seventeen cases of the Morax-Axenfeld type and in twenty-one of the Petit type.

Diplobacillary infections usually run a chronic course, the Morax-Axenfeld bacillus being associated with a chronic angular conjunctivitis or with a chronic blepharo-conjunctivitis, and the Petit bacillus with similar infections and also with cases of superficial serpiginous hypopyon-keratitis. Occasionally they give rise to much more acute conditions, for I have obtained diplobacilli on two occasions from cases of acute suppurative conjunctivitis clinically diagnosed as, and indistinguishable from, gonorrhœal ophthalmia, but in which no gonococci could be discovered.

On the other hand, the infection may be so mild and set up so little disturbance that it escapes recognition, and on several occasions I have obtained cultures of diplobacilli from apparently normal conjunctivæ from which cultures were taken as a routine procedure for prophylactic purposes prior to operation involving a perforating wound of the globe.

Although the diplobacillus may be, and often is, found alone in the conjunctival sac this is not invariably the case, for I have found associated with it one or more (and often several) of the following organisms, namely, *Bacillus xerosis*, *Staphylococcus albus*, *Micrococcus catarrhalis*, Bacillus of Koch-Weeks, and *Sarcina lutea*.

As regards its distribution in the body, it was believed prior to 1897 that diplobacilli were only to be found in the conjunctival sac. In that year Briand published his Paris thesis pointing out that they could often be found in the nose, even in the absence of any sign of conjunctival infection, and his results have been abundantly confirmed by Erdmann, Treacher Collins, and Gifford. In the majority of these cases the nasal mucous membrane was quite healthy, but in a few of them a condition of chronic rhinitis was observed. Erdmann, moreover, proved the virulence of diplobacilli present in the nose, for implanting nasal secretion containing them into the healthy conjunctival sac, proved by cultures to be free from diplobacilli, set up typical angular conjunctivitis swarming with diplobacilli.

Biard considered that the diplobacilli could produce infection of the conjunctiva and he believed that this occurred by way of the lachrymal duct, although Erdmann, while confirming his results, believed it to occur more frequently through infection by handkerchiefs, fingers, etc.

Hitherto extra-ocular distribution of diplobacilli has been confined to the nose, and I am unable to find any mention of it being found in any other situation. In the following case the diplobacillus of Petit was obtained in cultures taken from the throat, and I have therefore thought it worth placing on record.

Corporal E., 4th Grenadier Guards, reported sick on July 6, 1915, complaining of sore throat, with difficulty of swallowing and some stiffness about the front of the neck. There was an intense diffuse redness of the whole pharynx, with a little purulent exudation on the left tonsil, but no sign of membrane could be detected. There was also slight swelling and tenderness of the glands beneath the angles

of the lower jaw. He was sent to the Laboratory at the Queen Alexandra Military Hospital, Millbank, for a bacterioscopic examination of the throat. Swabs were taken and sloped blood serum tubes were inoculated. A gargle of chlorate of potash was ordered. On the following day growth on the blood serum had produced liquefaction with the characteristic pitting, stained preparations showing the presence of typical short, stout Gram-negative diplobacilli in almost pure culture, the only other organisms present being a few Gram-positive micrococci. No Klebs-Löffler bacilli could be found. Attention was now directed to the conjunctivæ, both of which were normal, being quite free from any trace of inflammation, and this was confirmed by Temporary Lieutenant D. Heron, R.A.M.C., Ophthalmologist to the Hospital. Swabs were taken from both conjunctivæ and inoculated on to blood serum, but no pitting resulted. Swabs were also taken from both nasal cavities and similarly treated, and diplobacilli were obtained from the left nostril, but not from the right.

Subcultures on agar from the pitted serum showed a copious greyish-white growth, which microscopic examination showed to consist of staphylococci mixed with large numbers of Gram-negative diplobacilli. By plating on serum the diplobacillus was obtained in pure culture. It was then found to grow readily on agar, producing a yellowish-grey confluent somewhat translucent growth. Growths also occurred on gelatine at 20° C. with liquefaction of the medium.

Two days later the pharyngitis was much less intense, but diplobacilli were again obtained from both the throat and left nostril. A gargle of zinc sulphate (gr. 1, ad. 1 oz.) was now given with instructions to irrigate the nasal cavities. Two days later cultures taken from swabs showed that the diplobacilli had disappeared from the throat but were still present in the nose, and the same findings were obtained on July 17, when the patient returned to duty.

It would thus appear that the pharyngeal infection was a staphylococcal one and that the diplobacilli were not the causal organisms seeing that they still persisted after the pharyngitis had cleared up.

It was also pointed out how speedily the diplobacilli were banished from the throat after a gargle of zinc sulphate had been given, and how persistently they remained in the nose, probably as the result of the difficulty experienced by the patient in carrying out the nasal irrigation with the zinc sulphate lotion.

Finally, considering the absence of diplobacilli from the conjunctival sac, their ready disappearance from the throat and their persistence in the nasal cavities lined by healthy mucosæ, we may conclude that this patient is a chronic carrier of the diplobacillus of Petit in the nasal fossæ and that the diplobacilli obtained from the throat were derived from this source.

UNIVERSAL ARM SUSPENSION.

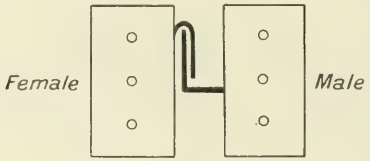
By Major M. SINCLAIR.
Royal Army Medical Corps.

This is composed of a movable wooden top and a fixed wooden perpendicular.

The lower perpendicular pole, L.P. (2 in. by 2½ in.), 5 ft. in length, is fastened to the top of the bed by means of a flat bit of timber, F (4 in. by 1 in.), 2 ft. 4 in. long, and bolted with two bolts to the pole with the bed headrail between them on the extreme right or left of bed.

The pole, L.P., has two sets of hinges, anterior and posterior, to enable it to be used for right or left arm, anterior for left and posterior for right. These hinges are set at 15 in. apart, the upper hinge being 4 in. from the top of pole, L.P. They also allow the top to swing 180°.

The hinges are composed of two parts—

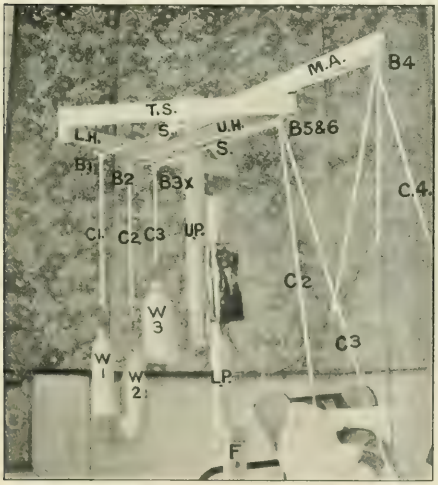


Hinge.

thus allowing the top to be easily moved, when the suspension is required for right or left arm as the case may be. By this method two male parts are fixed to the upper perpendicular, U.P., and four female parts to the lower perpendicular, L.P. In alternate suspensions, male and female parts are reversed. The top has a perpendicular part, U.P., and carries two fixed horizontals, L.H. and U.H., and a movable arm, M.A., which is hinged to the upper perpendicular, U.P. This top can be completely removed from the pole by pushing the top upwards, and thus separating the male from the female hinges. This renders it applicable to right or left arm. The upper perpendicular, U.P., is made of the same material as the lower fixed perpendicular, L.P. (viz. 2½ in. by 2 in.) and is 3 ft. 2 in. in length. The two fixed horizontals, L.H. and U.H., are set at right angles one above the other. They are 2 ft. 8 in. in length, the material is 3 in. by 1 in. They extend 2 ft. in one direction and 8 in. in the other.

The upper U.H. rests on the horizontal L.H., which in turn rests on a block of wood, X, all three being screwed to the upper perpendicular, U.P. From the end of the shorter pieces of these two horizontals, two stays, S. (2 in. by 1 in.), are fixed to the top of the upper perpendicular to counteract the weight that has to be applied to the longer ends. No

Showing arm suspension applied.



Arm suspension, showing details.

dovetailing is done so far to avoid any weakening of the superstructure. The ends of the longer pieces of the two horizontals are joined by a piece of wood, T.S. (3 in. by 1 in.), and, in order to keep this level, it is let in at the end which is in contact with the higher of the two horizontals and rests on the top of the other horizontal. At each end of this transverse stay, T.S., is a flat iron upright (4½ in. by 1 in. by ⅛ in.) projecting upwards to act as a check, K., to the movable arm and at the same time give more strength to these joints.

The movable arm, M.A. (3 ft. by 1 in.), is hinged to the upper perpendicular, and the upper edge 4 in. from the top of U.P. moves through an angle of 90°. It is for the attachment of a block when the arm is extended, as in an arm "Thomas" splint. When the arm is extended (as in the photograph) in an arm "Thomas," there are three eyes of wire fixed to the splint, two at the ring (one on each side of the iron bars of the splint), and the third at the middle of the transverse bar at the hand end. Three cords, C1, 2, 3, are attached to these eyes—watch-chain hooks are very convenient—and each passes through its respective two blocks, while a suitable weight is attached to the other end.

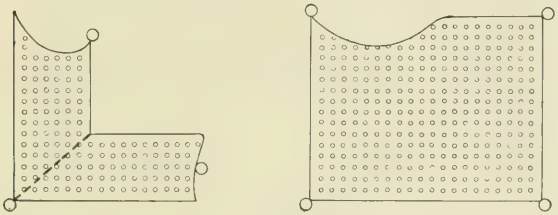
For a right arm the cord C1 from the hand end passes through block B4 which is attached to the end of the M.A., then passes to block B1, which is fixed to horizontal L.H.

It has been found since this photogtaph was taken that block B1 is better fixed at the other end of lower horizontal L.H., so as to immobilize the arm, M.A., and a weight (sandbag) of about 2 lb. is attached at the other end. The cord C2 is attached to the outer eye at the ring end of the splint, passes through block B5 which is fixed at the junction of T.S. and U.H., then through block B2 on L.H. and carries a weight of about 4 lb. Cord C3 passes from the inner eye of the splint through the other block B6, fixed at the junction of T.S. and U.H., thence to block B3 on L.H. and carries a weight of about 6 lb. The blocks B1, 2, 3, should be so placed on L.H. as to allow free play of the weights without touching.

In the accompanying photographs, the irrigator rubber tube and two cords supporting the tubing which is leading to a wound that is being treated by continuous irrigation are not lettered to avoid confusion. A gutter of perforated zinc is fixed to the sides of the "Thomas," and holes are burned in the zinc opposite any drainage-tubes. To prevent tissues herniating through these exits, the holes should not be too large. The whole is sterilized by flaming, and the arm with wounds is laid directly on this bed of zinc.

This form of suspension of the splint conforms to all permissible movements of the limb, and is a great comfort to the patient, who is free to move at his will or to sit up by his bedside if he wishes. Wounds so treated, drained freely, are readily dressed and are open to inspection at all times.

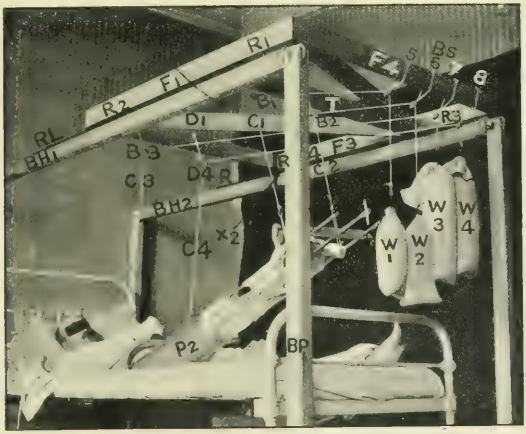
By redistribution of the weights and blocks this suspension can be adapted to a rectangular wire splint or a square wire frame, both of which are filled in with perforated zinc, for use with the flexed elbow.



Wire splint.

UNIVERSAL LEG SUSPENSION.

The suspension consists of a fixed frame carrying a travelling cradle from which the limb in its splint is suspended by a system of weights and pulleys.



Leg suspension applied. P.z., perforated zinc.

Frame.—Material, 3 in. by 2 in.

A length of T section iron rail is screwed to the upper surface of an 8-ft. wooden bar for a distance 5 ft. 6 in. from the foot end.

Two such bars are supported at their extremities by 6-ft. uprights placed so that the rails are 3 ft. 3 in. apart and parallel to each other.

The uprights are firmly fixed to the floor by suitable stays, and the pair at the head end are joined by a transverse bar.

Cradle.—Material, 3 in. by 1 in.

All dimensions are outside measurements.

The travelling cradle is rectangular with sides F1 and F3, 4 ft. in length, carrying two window-sash pulleys, each let in 4 in. from the extremity R1, 2, 3, 4.

The ends F2 and 4 are 3 ft. 4 in. in length.

This rectangle is divided by a transverse stay T. at 1 ft. 5 in., and the two smaller rectangles thus formed have diagonal stays, D1 and 2, to prevent tacking and give rigidity. On F2 and T. iron checks, X2 and 1, (6 in. by 1 in. by $\frac{1}{8}$ in.) project vertically downwards, just clearing the horizontals B.H.1 and 2. Strong 2-in. screws are fixed half-in into the inner sides of B.H.1 and 2 to engage with these checks.

These are best adjusted experimentally. Sixteen round-headed 2-in. screws are fixed half-in into the upper edges of F2, T. and F4 in the following way (diagram of suspension). On F2 four screws at the sixth and fifteenth inch from each side.

On T. four screws at the second and eleventh inch from each side.

On F4, eight screws at the fourth, ninth, fourteenth, nineteenth, twenty-first, twenty-sixth, thirty-first and thirty-sixth inch.

There are now sufficient for a right and left leg and are for the attachment of the single blocks of the Hodgen's splint ($1\frac{1}{2}$-in. block is the best size).

In slinging a right fractured femur in a Wallace's splint, as in photograph and figure, blocks are attached to screw S1, 2, 5, 6, 13, 14, 15, 16.

There are 4 eyes of wire fixed to the splint: 2 on the ring (1 on each side below the bars of the splint), and the other 2 to the transverse bar carrying the foot piece.

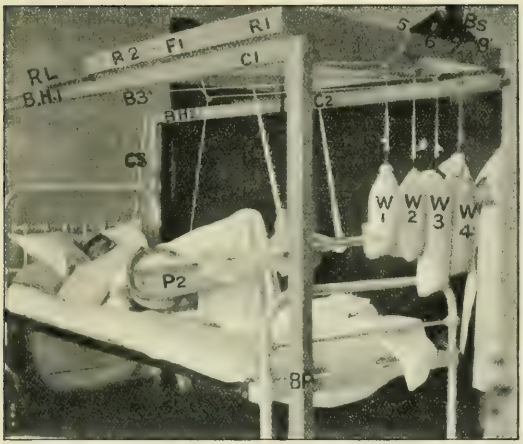
Four cords, C1, 2, 3, 4, are attached to these eyes—watch-chain hooks are very convenient—and each passes through its respective two blocks, while a suitable weight is attached to the other end.

Cord C1 from outer foot attachment passes through block B1 which is attached to screw S5 on T., then through block B5, which is attached to screw S13, and carries a weight of about 7 lb.

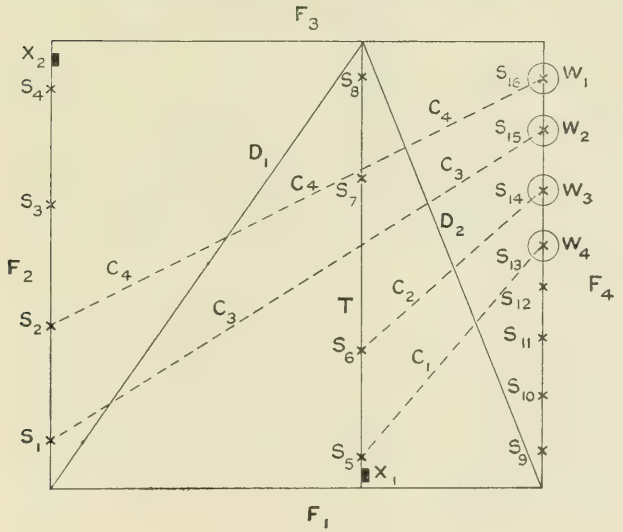
Cord C2, from inner foot attachment, passes through B2, which is attached to screw F6 on T., then through block B6, which is attached to screw S14, and carries a weight of about 7 lb.

Cord C3 from outer ring attachment passes through block B3, which is attached to screw S1, then through block B7, which is attached to screw S15, and carries a weight of about 14 lb.

Cord C4, from inner ring attachment, passes through block B4,



Showing patient raising pelvis unaided four days after injury.



9 Diagram of suspension.

which is attached to screw S2, then through block B8, which is attached to screw S16, and carries a weight of about 14 lb.

This suspension allows the patient very free movement, is a great comfort to him, and nursing is made comparatively simple.

The second photograph shows the patient, who has a fractured femur and ulna, raising his pelvis unaided four days after injury.

REMOVAL OF A SHRAPNEL TIME-FUSE FROM THE LEFT SHOULDER. DESCRIPTION OF A SPLINT.

By Lieutenant A. E. MORTIMER WOOLF.
Royal Army Medical Corps.

In removing foreign bodies from various parts of the body, I have often noticed the rarity of finding a piece of shell of any considerable size. Quite large wounds may be caused by a projectile of astonishingly small dimensions. Previously to this case the largest piece of shell I have removed had penetrated the knee-joint, completely severing the internal condyle of the femur from the rest of the bone. In this case the portion of shell did not measure one cubic inch. The lodgment of a complete shrapnel time-fuse in almost perfect condition is an event of such rare occurrence that it seems to be worth while placing it on record.

The patient was wounded on May 25, at about 2.30 p.m. He is a stretcher bearer and was going up to the trenches to collect wounded. He advanced to within about four yards of his destination when he heard the shriek of a shell approaching. He turned his back on the trenches and almost immediately felt a severe blow on the back of the left shoulder. The blow did not knock him down, but caused him to stagger and then, as he somewhat naively expressed himself, "it set me running." He reached a ditch about one hundred yards away, where he fell down and in about twenty minutes was removed to the dressing station, where his wound was dressed and painted with iodine. That night he was taken to the clearing station, where, under an anæsthetic, a tube was inserted. On the following day he was sent to another clearing station and then despatched on the train, arriving at a General Hospital at the base three days after the reception of the original injury.

On admission, the patient was in considerable pain, much more so than is usually the case. There was a wound over the posterior border of the deltoid about one and a half inches long, through which a tube had been inserted. The other end of the tube appeared through a much smaller wound situated over the pectoro-deltoid interval. This wound looked like an operation incision, and the patient thinks was not caused by the original injury.

Owing to the severity of the pain he was taken to the theatre almost immediately. On inserting my finger into the posterior wound, I encountered a round smooth object which for the moment I thought was the head of the humerus, dislocated and externally rotated. On retracting the edges of the wound, I saw the piece of brass at the top of the round part of the time-fuse, and immediately recognized the cause of the trouble. The fuse was tightly impacted and the greatest convexity was situated about three-quarters of an inch from the surface. The wound had to be enlarged considerably to allow of removal which was

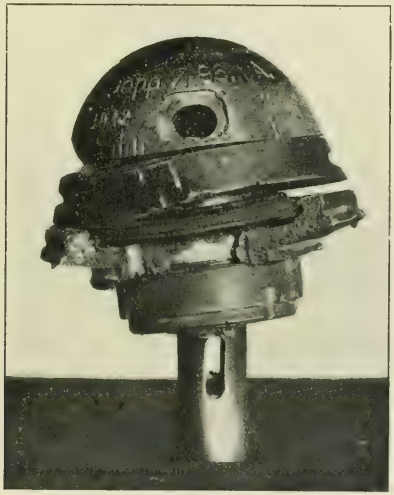


Fig. 1.

accomplished without difficulty. There was a comminuted fracture of the humerus below the surgical neck; some loose fragments of bone were removed and the sharp pointed ends of the upper and lower fragments were excised with bone-cutting forceps; drainage was provided for.

The fuse was lying with the stem against the bone. The fracture, though a severe one, was not nearly so comminuted as that caused by many a smaller piece of shell. On removal there were septic pieces of muscle attached in the cracks of the split metal and the odour was most revolting. The photographs show two views of the specimen in nearly

perfect condition. The stem of the fuse was lying nearly horizontal, with a slight inclination downwards. I suspected that the injury was due to a ricochet, but on questioning the patient afterwards, he was unable to throw any light on this question. The weight of the fuse is ten ounces.

In the treatment of compound fractures of the upper end of the humerus due to projectiles two difficulties are encountered:—

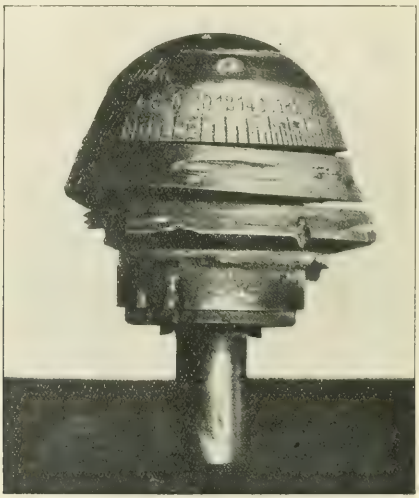


Fig. 2.

(1) The application of a retentive apparatus.

(2) The dressing of the wound, without it being necessary to remove the splint.

For fractures below the shoulder the modified Thomas's splint is excellent and fulfils both these requirements, at the same time allowing the lower fragment to be placed in any degree of abduction necessary. It is unfortunately not applicable to wounds high up as the circular band interferes with the dressing. In order to obviate this, a splint of the Thomas's variety but modified, I believe by Mr. Robert Jones, has been supplied, with the upper collar so made that it fits closely to the neck, and so gives access to the wound. The one drawback to this splint is

that it is impossible to abduct the lower fragment and at the same time prevent internal rotation.

In order to obviate these difficulties I have adopted the following plan: A piece of perforated zinc plating, measuring 23 in. by 8½ in., and supplied in the Army fracture boxes, is cut for about 1½ in. on both sides, and in two places, one about 7½ in. from the end of the plate, and the second about 6 in. from the other end. The plate so cut is bent as

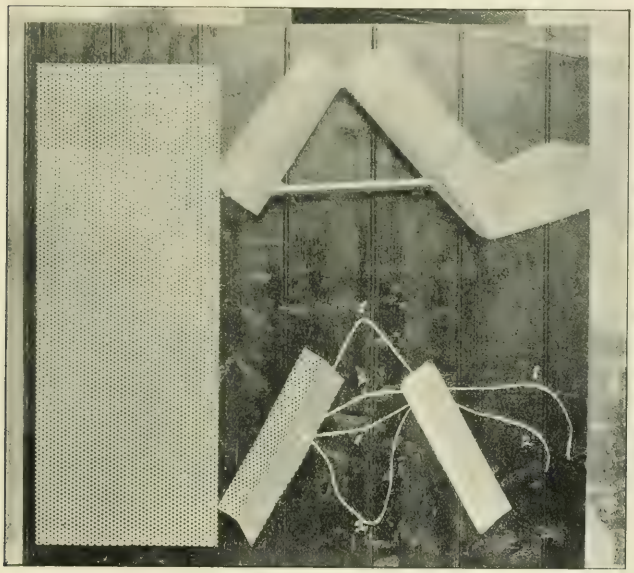


Fig. 3.

shown in the photograph, so that a thoracic piece, arm piece and forearm piece are formed. The position of the actual cuts should be determined roughly by measuring of the patient.

The cuts are easily made by any strong pair of shears, such as plaster of Paris shears or even a strong pair of scissors. The very flexible plating is now moulded to the shape of the parts to which it is to be applied, and is cut away where it interferes with the subsequent dressing. The sharp corners are rounded off, and rigidity is obtained by the following methods:—

(*i*) Two holes are bored at each end of a piece of wood about seven and a half inches by two and a half inches. A piece of string is threaded through the two holes, passed through two opposite holes in the zinc plating, the ends crossed over and threaded back again through the zinc plating into the holes in the wood. By this means, the string end which was originally threaded through the right-hand hole in the wood, finally emerges through the left-hand hole. The wood is then firmly fixed by tying at both ends. The distance between the middle of the wood and the apex of the axillary piece should be about four inches, so as to allow

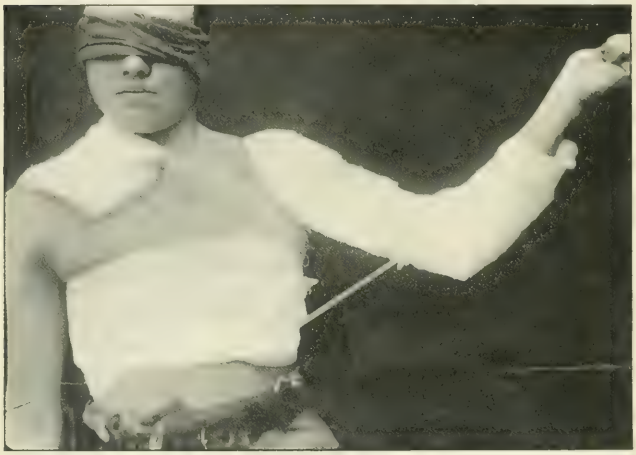


Fig. 4.

a bandage to be passed easily through this space. The forearm piece which is bent up will allow holes in the plating of this portion to come into opposition with holes in the arm piece, and string is threaded through in precisely the same way as described above, on each side, in order to secure rigidity between the two pieces.

(*ii*) The thoracic piece should be bandaged to the chest separately, and before starting to bandage the arm. The arm is bandaged over the highest point of the shoulder and under the axillary piece and then down the arm and forearm. In dressing the case, it is only the latter bandage that is removed ; the bandage fixing the thoracic piece to the chest keeps the splint in position while the dressing is carried out. Extra security

can be obtained by passing a triangular bandage under the axillary piece and tying over the shoulder of the opposite side, as shown in the photograph.

I have now used this splint on twelve cases and have found it very satisfactory. It is easily applied while the patient is under the anæsthetic, and takes about fifteen to twenty minutes to complete.

By making each splint at the time of operation, the individual requirements of a particular case are catered for. After the operation the patient is quite comfortable, rigidity is adequate, and subsequent dressing much facilitated.

A PORTABLE "BED-REST" STRETCHER.

By Major ERNEST FINCH.
Royal Army Medical Corps (T.F.)

The desirability of placing men, suffering from wounds in the chest and abdomen, in a sitting position, is well recognized. The "bed-rest" attitude is also adopted in treating diseases of the chest, and men suffering from the effects of asphyxiating gas. Another point to be emphasized is, that this sitting posture should be maintained while the patient is being moved from one place to another, in the same room, building, or by convoy.

With ordinary regulation (Mark II) stretchers this may be done by devices, such as a pile of blankets, a box, or some other equally inconvenient makeshift.

The problem presenting itself was, then, the construction of a portable stretcher on the bed-rest principle.

This has been solved for me by Lieutenant B. G. Bouwens, A.S.C., M.T., who is in charge of a workshop unit at the front. It will carry a patient in any position, from lying flat to sitting up, either by bearer or in an ambulance. There is no mechanism which can get out of order, or stick up with mud, rust, or misuse.

The poles of the bed-rest are triangular in shape, and so placed that with the foot-rest it is improbable that a patient will roll off. The main strain on the canvas will be beneath the buttocks; an extra strip of canvas is placed here, which adds to the durability of the stretcher. The foot-piece is adjustable, so that the stretcher can be used for a short man as easily as for a tall one.

The following is a brief description:—

Parallel to the main poles of the stretcher are hinged two pieces of wood, forming the poles of the rest, triangular in shape, two feet three inches in length.

The canvas is fixed in its upper part to the hinged poles of the "rest," and in its lower part to the main poles of the stretcher. The upper part

The stretcher closed: a strap secures the bed-rest to the poles.

The stretcher prepared for a lying down case.

The stretcher prepared for a case for which the bed-rest is required.

is therefore capable of being set at a varying angle to the lower, without interfering with the carrying position of the stretcher poles, which are permanently horizontal.

Adjustable metal props are provided, to vary the inclination of the bed-rest, and, when closed, fold down just clear of the carrying handles. The two hinged poles are kept open by a traverse bar.

The foot-rest consists of curved iron uprights, kept open by a traverse bar. Canvas is stretched between them. The uprights are shaped to engage with pins in a rack when open, so that the position can be adjusted to the height of the patient. The whole foot-rest can be folded flat, if an ordinary lying down case is on the stretcher.

There is no difficulty in completing "close stretcher."

The cost of converting an ordinary (Mark II) stretcher into the "bed-rest" variety works out at three francs a stretcher.

The weight is forty pounds, compared with thirty pounds which is the weight of an ordinary regulation stretcher.

It takes a little longer to open and place the stretcher in position than it does when working with the regulation type, but it folds up to about the same size.

In conclusion, I must thank the officers of the 3rd West Riding Field Ambulance for many suggestions, and Lieutenant Bouwens for the trouble and time he and his workshop unit have taken.

The value of the stretcher is undoubted, and already, in many cases, it has filled a much felt want, and has been a boon to wounded men in the field ambulance hospital.

Translation.

ON THE USE OF PORTABLE KITCHENS (DES CUISINES ROULANTES) AS APPARATUS FOR DOUCHES AND STEAM DISINFECTION IN THE FIGHTING LINE AND IN ARMY CORPS (DANS LES SERVICES DE L'AVANT ET LES CORPS DE TROUPES).

By M. le Dr. CHAUCHARD.

Typhus and recurrent fever (la fièvre récurrente) have at all times been dreaded scourges of armies in the field. We know that they are spread by lice. Many means of destroying lice have been advocated; the only one of indisputable value is the steam stoving of clothing and linen, together with and at the same time as bodily cleansing by hot soapy douches, followed by the rubbing in of antiseptics. The construction of steam stoves in regiments in the fighting line appears, at first sight, practically impossible, and so this process is not used. However, all army corps have at hand first-class apparatus, admitting the establishment, without expense, and in a simple and practical way, of a service of douches and disinfection. This apparatus is the portable kitchen.

As Senior Medical Officer (*Medicin-chef*) of a regiment in the first line, I am using with complete success this means of disinfection, and that by adapting to the portable kitchens an arrangement small in cost and within the reach of all.

(1) *The Steam Stove.*—At the disconnecting pipe (or at the safety valve, previously unscrewed) of one of the boilers an elbowed pipe is fixed. The free end of this passes through an opening in the upper part of one of the staves of a large barrel (those which I use have a capacity of five hundred litres). The upper end of this barrel has been taken off, and made into a lid, on the lower side of which are hooks on which the clothes are hung. This lid is raised by means of a pulley. The lower end of the barrel is pierced with holes for the escape of air and the exit of steam. The boiler is half filled with water and heated by coal or coke. The steam is conducted by the elbowed pipe into the barrel, of which it fills the upper part first, as its density is less than that of the air. Little by little, in proportion as the pressure increases, it drives the air, after the manner of a piston, towards the lower part of the barrel. Thus, in a few minutes, the air is completely driven out. At this moment the thermometer placed in the interior of the lower part of the receptacle rises to 100° C., and remains there as long as the steam is given off. The parasites and their nits are destroyed in fifteen minutes from this time.

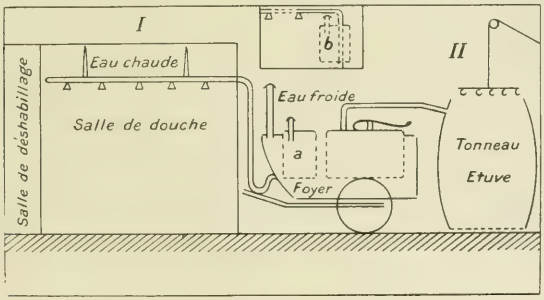
This apparatus provides for the stoving of the clothes and linen of one hundred men per hour. Failing a barrel, a wooden case could be arranged covered with sheets of zinc or light sheet iron.

(2) *Douche Installation.*—There are two types of portable kitchens:—

(*a*) Boilers with outlet tap at the bottom and with covers fastened at the centre by means of a lever and a spiral spring.

(*b*) Boilers without a lower tap and with covers fixed by means of bolts (autoclave system).

First type; boiler with outlet tap (scheme *a*). The cold water comes in by the disconnecting pipe; this water is supplied either by a pre-existent system of pipes, or from a barrel raised up (four to five metres), acting as a reservoir. A hose of canvas or rubber is fitted to the lower outlet tap, and at its other extremity there is fixed an iron or lead pipe bearing every eighty centimetres a watering rose (total, five on the apparatus I set up). The spiral spring is blocked by means of two wooden wedges to stop escapes between the cover and the boiler.



Second type (scheme *b*). The water comes in in the same way. To let it out a hole has been made in the cover which permits the introduction, down to the bottom of the receptacle, of an outlet tube for the water, which is connected with the pipe carrying the roses by means of a hose of canvas or rubber.

The boiler is filled with water and heated. In a few minutes a continuous flow of hot water is obtained, the temperature of which is regulated as required by opening the inlet tap more or less, or by varying the intensity of the heat of the fire.

Two men take their douche under each rose, soaping one another. Each douching takes five minutes, so we douche more than one hundred men per hour.

To make this apparatus there is needed: A tap for the inlet of the water, six metres of iron or lead piping, five roses, one and a half metres of rubber or canvas hose, a barrel, and, in the type of boiler without a tap, there is required besides an adjustment for the opening pierced in the lid. The cost of all these fittings is negligible, considering that the

allowances (les circulaires allouent) to army corps for douches is 45 centimes per man per month, which, for a regiment of three thousand men gives a monthly credit of 1,350 francs.

These fittings are set up in a large room (une salle) or hut, six metres by four, in one corner of which an undressing closet has been arranged by a boarded partition; the "kitchen" remains outside. In cases where the ground is not cemented it will do to make an open flooring over light beams, which would permit the water to run away.

Up to the present I have installed five stations, one of which is working in a ravine, sheltered from artillery fire, about five hundred metres behind our trenches. The officers and men of the companies in the first line are its assiduous clients. To the others there come, besides the men of the reserve companies of my regiment, men from neighbouring regiments; four regiments of infantry, artillery, army service corps (du train des équipages), engineers, etc., the German prisoners of the Division; in all more than ten thousand men.

Two men suffice for the service of each of these stations; a stoker, capable, in case of need, of doing soldering, and an orderly (stover) taken from a resting company.

Each infantry regiment has four portable kitchens, two to each battalion, the regiment consisting of two or three battalions. It is thus easy to arrange among them a sanitary service which will fit in with the cooks' hours, as setting up and taking down the apparatus only occupies the work of a few minutes.

Current Literature.

The Ætiology of Typhus Exanthematicus (H. Plotz, P. K. Olitsky and G. Baehr. *The Journal of Infectious Diseases*, vol. xvii, No. 1, July, 1915).—This paper is divided into three parts: (1) Bacteriological studies; (2) serological studies; and (3) experimental studies.

The first part of the paper deals with the work which led up to the discovery by Plotz of the organism of typhus fever. The epidemic form of typhus is characterized by its comparatively mild course, and low mortality. This disease is known in America as Brill's disease. Louria and Friedman have published papers pointing out the resemblance between this disease and cases of mild typhus which they had seen in Europe.

Striking cross-immunity experiments have been described which show that a cross-immunity exists between monkeys infected with Brill's disease and those infected with Mexican typhus.

In the search for the ætiology of typhus fever one of the most important aids was the fact that the virus of typhus had been found in the circulating blood during the febrile period of the disease. The work of Nicolle, Ricketts and Wilder, Gavino and Girard, and of Anderson and Goldberger, all pointed to the fact that the virus of typhus is not filterable.

Ricketts and Wilder described a bacillus which they saw in blood films stained with Giemsa's stain. Plotz says that this bacillus bears a certain morphological resemblance to the organism which he has cultivated.

It has been surmised that since typhus had been proved to be an insect-borne disease, a protozoan origin would be found, but Ricketts and Wilder called attention to the fact that the rat-flea carries the bacillus of plague, as does the tick the spirillum of African tick fever.

The same authors also pointed out that typhus is an acute, self-limited disease, and that one attack confers immunity, a feature characteristic of many bacterial diseases but extremely unusual in protozoal infections.

These facts led to many attempts to cultivate the virus from the blood in cases of typhus fever, and for this purpose aerobic methods were used with uniformly negative results. The media used were plates of agar and glucose agar (often with ascitic fluid added), flasks of broth, and two per cent glucose broth with and without ascitic fluid.

Plotz not only repeated the aerobic methods, but also used anaerobic methods of cultivation. His method of culturing was as follows :—

Fifteen cubic centimetres of blood are drawn from a vein in the antecubital space with the usual aseptic precautions. For the aerobic method he poured two plates of agar and two of glucose agar. The remainder of the blood was distributed in one or more flasks (one hundred centimetres) of broth, and two per cent glucose broth. The agar used was of two to three per cent concentration, prepared with Liebig's meat extract, and of 0·9 to 1·1 per cent acidity. Two cubic centimetres of blood were added after the agar had been melted and cooled to 40° C. It was well mixed and the plates poured. Rabinowitsch's method, consisting of five cubic centimetres of blood added to one hundred cubic centimetres of equal parts of broth and ascitic fluid with four per cent glycerine added, was also tried.

These all gave negative results, although four of the cases from which blood had been taken afterwards gave a positive result with the anaerobic method of culture.

Plotz in the early part of this study tried many methods of anaerobic cultures which failed. The first successful results were obtained by the use of the ascitic fluid, kidney tissue, and liquid paraffin medium of Noguchi. By this method he obtained the organism in two cases of epidemic, and in one case of endemic typhus fever. The method of Liborius-Veillon proved more satisfactory — modified in that serum glucose agar was used instead of glucose agar. Plotz uses tubes fifteen centimetres long by two centimetres wide, each containing about twenty cubic centimetres of two per cent glucose agar. The agar is made in the same way as for the aerobic method of cultivation. Eight tubes are used for each blood culture. The medium is melted and cooled to 40° C., and into each tube three cubic centimetres of blood are injected directly from the syringe and to the mixture four cubic centimetres of ascitic fluid are added. The contents are mixed by pouring from one sterile tube into another, care being taken not to introduce any air bubbles. The contents are allowed to cool at room temperature, or better still by immersion in ice-water, and then a layer of plain agar

two centimetres deep is added to each tube, The tubes are then incubated at 37·5° C.

The success of the culture depends greatly on the selection of the serum. It was found that only such sera should be used as are bile-free and have a specific gravity of 1015 or over.

The sterility of the ascitic fluid must be ascertained by aerobic and anaerobic culture.

Colonies appear in from three to sixteen days. They can be removed from the tube by breaking it across, and subculturing into slopes of 0·5 per cent glucose serum agar (a quarter the volume of serum), and placing the inoculated tubes in Buchner tubes.

The colonies usually appear in the lower two or three centimetres of the tubes. Each colony first appears as a small opaque spot; as it grows a brown area of precipitation develops in the media about it. The colony itself varies from one to six millimetres in diameter (including the area of precipitation). In cross-section it is Y-shaped, brownish in appearance, and soft in consistency. The arms of the Y are fusiform.

The organism is a small pleomorphic, Gram-positive bacillus, non-motile, not capsulated, not acid-fast, and does not form spores. Its length is from 0·9 to 1·93 μ, breadth from one-fifth to three-fifths of its length. Most of the organisms are straight though occasional ones are slightly curved. Coccoid forms also occur. Degeneration and involution forms appear early.

As to its cultural characteristics besides the usual cultures in Buchner tubes, a variety of media in long tubes (20 cm. long by 1·25 cm. wide) have been used. The tubes were inoculated with a saline emulsion, and incubated at 37° C. for twenty days. The best growth was obtained in two per cent glucose agar with ascitic fluid added, the growth extending up the tube to within 2 to 2½ cm. of the top of the medium. Precipitation takes place after the growth becomes profuse.

On potato, after four days, an invisible white growth forms, which is demonstrable on scraping the medium. There is no growth on gelatine. The organism produces acid in glucose, maltose, galactose, and inulin with precipitation, but no visible gas is formed. There is no change in raffinose, mannite, arabinose, saccharose, dextrin, or lactose. It produces slight acidity in milk in eighteen days. Superficial colonies when they appear on plates, which is not often, are opaque, 1·5 to 2 mm. in diameter, with fading, irregular edges. Each colony is surrounded by a zone of fine, dust-like dots, localized in an area of precipitation. The colonies are cream by reflected light and opaque by transmitted light. The deeper colonies vary in size from a pin-point to 1 mm. In shape they are round, oblong, or triangular, the last shape prevailing.

It is worth noting that Plotz says that for keeping the organism alive 0·5 per cent glucose agar is used, as the larger glucose content is presumed to decrease the virulence to a greater extent. The organism is an obligatory anaerobe. Its thermal death-point is 55° C. for ten minutes, which coincides with the thermal death-point of the virus that Anderson and Goldberger demonstrated in the blood of typhus patients. An emulsion of the organism when passed through a Berkefeld filter, size N, fails to produce any growth in 0·5 per cent glucose serum agar, while a control of the emulsion not so passed does so.

The general result of blood cultures was as follows:—

In epidemic cases, the blood of seven cases being cultured, the organism was recovered from all in pure culture, during the febrile stage. In endemic cases the bacillus was recovered in fifty-three per cent. The cultures were taken on varying days of the disease up to the day of the crisis.

Blood cultures were made in six of the epidemic cases after the crisis, and all proved negative. In nine endemic cases cultured after the crisis two yielded the bacillus, one twelve hours and the other thirty-six hours after the crisis.

The authors of this paper also made anaerobic cultures from one hundred and ninety-eight other febrile cases. These represented a variety of conditions such as typhoid, acute osteomyelitis, acute rheumatic endocarditis, chorea, acute nephritis, influenza, liver abscess, splenic anæmia, etc. In none of these cases was an organism recovered in any way resembling the bacillus isolated from cases of typhus fever.

As to the day of disease on which the most marked bacteriæmia was present, it was found to be impossible to determine the day of onset in the patients from whom blood was taken. It was, however, found that the best results were obtained four or five days before the crisis. In cultures taken from the blood of inoculated animals the organism was most frequently found when the blood was taken early in the disease. This probably holds good in man also.

There seems to be a marked relation between the amount of bacteriæmia present and the severity of the disease. The bacillus was found eighteen times as abundantly in the epidemic as in the blood of the endemic cases.

The serological studies are dealt with by Olitsky. He discovered the presence of complement-fixing antibodies against the isolated bacillus only in the cases of typhus fever, and in no other diseases. After some preliminary experiments he used an antigen consisting of the filtrate of a killed, autolyzed emulsion of the bacillus of typhus, using a mixed emulsion of both the epidemic and endemic strains.

Eleven cases of epidemic and 34 cases of endemic typhus were tested. Nine of the epidemic and 30 of the endemic were tested after the crisis. Of these the total number of positives was 28, or 71·8 per cent. Of all the cases tested irrespective of the stage of the disease the total positive results were 29, or 64·4 per cent.

At the height of the disease 25 cases were tested, and only 2 positive results were obtained. Ten cases were tested at the crisis, and 4 were returned as positive; hence we see a tendency towards an increased number of complement-fixing antibodies at the crisis. All the controls gave negative results, and covered a number of acute febrile diseases. For agglutination estimation he uses the microscopic method rather than the macroscopic, for these reasons :—

Large quantities of the bacilli necessary for suspensions as agglutinogens are difficult to obtain, owing to the sparse growth of organisms and the strict anaerobic conditions required. The bacillus even without the addition of serum has a marked tendency to clump on standing. Also, where there is a positive result with the macroscopic method there is always a positive one with the microscope, and this method conserves the serum.

The serum used is inactivated at 56° C. for half an hour. The

final dilutions used are 1 in 20, 1 in 50, 1 in 100, 1 in 200, 1 in 400. The tests are read after one hour at room temperature.

The average titre of typhus fever serum is 1 in 200.

At first the two strains of bacilli were used and a cross-agglutination was observed. Later a mixed agglutinogen was used, and when this was employed positive agglutinations were obtained in more cases than positive complement-fixations. In 43 typhus cases (including the epidemic form), during some period of the disease, 39, or 90·7 per cent. were positive. Just as in complement-fixations, the agglutinations were usually absent at the height of the disease, increased in amount at the crisis, until they reached their maximum well on in the apyrexial period. They can be demonstrated at least five months after the crisis. Of 49 controls 7 showed agglutinins in dilutions of 1 in 20, but none in higher dilutions. These 7 cases consisted of the following: 1 was a brain tumour; 2, valvular heart defects; 2, general paralysis; 1, carcinoma of the tonsil; and 1 a case of alcoholism. In all complement-fixation was negative.

On this account Olitsky regards as positive only such cases as give agglutination in dilutions of 1 in 50 or higher. Agglutination with other than typhus organisms gave negative results in every case.

A considerable resemblance in the results was obtained in investigating precipitins to those obtained in the study of agglutinins and complement-fixation. No bacteriolysins were demonstrable. Opsonins are present. The index rises at the crisis and remains high in the convalescent stage of the disease. Olitsky considers that this method (opsonization) is one of the most potent, on the part of the patient, in overcoming the disease. He proved to his satisfaction by cross-fixation and cross-agglutination that the epidemic and endemic bacilli are merely two strains of the same organism. Experimental typhus in monkeys gives rise to serological changes in the same manner as in man. Of 6 monkeys that reacted to typhus virus, complement-fixation and agglutination tests were positive in 5, or 83·3 per cent. These reactions were still positive twenty days after the crisis. The average normal monkey has no such antibodies present. To produce these antibodies both in man and monkeys, large doses of antigen have to be given. In the case of guinea-pigs the result of inoculating the virus is quite different to that observed in the case of other animals. A guinea-pig injected with blood from a typhus patient on April 14, 1914, reacted on April 22. On June 16 it was again injected with a guinea-pig typhus virus. There was no reaction, hence the animal was proved to be immune. The animal was bled on July 7. Although the guinea-pig was proved immune, yet the serum contained no agglutinins or complement-fixing bodies. This experiment was repeated with other guinea-pigs.

Rabbits are not susceptible to the typhus bacillus in small doses. If given intravenously in large amounts they die in three to seven days.

The autopsy reveals no distinct pathological change. When small, yet increasing, doses of the bacilli are given, rabbits develop very potent immune serum, with especially high opsonic indices when compared with normal rabbits.

Observations made on the blood of two individuals proved very interesting. Both were definitely exposed to the typhus virus, one to

both types and one to the epidemic form only. At no time did either have any symptoms that could be recognized as typhus fever, yet they both developed complement-fixation bodies and agglutinins.

Previous work on the transmission of typhus had established the infectivity of typhus blood from man both for man and for animals.

The authors of this paper set out to ascertain whether the organism recovered from the blood of typhus patients was also found in the blood of animals inoculated with typhus blood. For this purpose, twenty-four guinea-pigs were inoculated with from 2·5 to 4 c.c. of defibrinated blood from patients with typhus fever diluted with an equal volume of normal salt solution.

Blood culture was made by cardiac puncture and grown in the usual media. A bacillus, morphologically and culturally identical with the one recovered from the blood of individuals with typhus fever, was recovered from the guinea-pig's blood in eight out of twenty-four, or in 33·3 per cent. They put down this low positive to the small amount of blood cultured—three cubic centimetres. The average number of colonies obtained from the eight positive bloods was three. This seems reasonable, as in man the positive results from fifteen cubic centimetres of blood produced only nine colonies in epidemic cases, and 2·06 in endemic cases. The febrile reaction at the time of the blood culture was proved to be due to typhus fever, as the blood of the guinea-pig which was the subject of the experiment was inoculated into other pigs, and all developed the disease after the usual incubation period of seven to fourteen days. They found that the best way of inoculating the guinea-pigs was by the intraperitoneal route.

Of eight guinea-pigs with positive blood cultures there was no doubt that the reaction was due to typhus, and in three guinea-pigs inoculated with blood which had given a negative result none developed the disease. Blood from six other animals with negative blood cultures was inoculated into thirteen guinea-pigs, and all developed the disease. This discrepancy with the blood culture may have been due to the small amount of blood used, as with human cases in which large quantities of blood were used no such discrepancy ever occurred.

Blood cultures were also made from four monkeys that had received an intraperitoneal inoculation of typhus virus. In one the blood culture was positive on the third day of the fever. The blood of this monkey produced the disease when inoculated into a guinea-pig. The relations between the blood culture and the severity of the disease can be noted both in animal experiments and in human cases; and is very suggestive that the bacillus is the cause of the disease. As to the relationship between the blood culture and the stage of the disease at which the blood is taken, it was found that the blood cultures taken twenty-four to seventy-two hours after the onset were positive in fifty per cent of animals. In the majority of the animals the fever reached its highest point on the second or third day. The finding of the bacilli in the blood of a guinea-pig twenty-four to thirty-six hours after the crisis was in accordance with the results of Nicolle and Conseil, and Anderson and Goldberger, who found the blood of a typhus case to be infective for animals twelve to thirty-two hours after the crisis.

It was found that the strains of the typhus bacillus when subcultured repeatedly, or when kept on artificial media for more than three or four

weeks, completely lose their virulence for animals. When inoculated into guinea-pigs only a transient rise of temperature was observed during the first twenty-four hours after the inoculation, apparently due to the action of the endotoxins.

This rise of temperature is completely done away with if the organisms are suspended for a half to one hour in a 1 in 20 to 1 in 100 dilution of typhus-immune serum.

On cultivation on artificial media for more than one month all strains lose this toxic action as well. In spite of repeated passage through mice and guinea-pigs it was found to be impossible to restore the virulence after it had once been lost.

Experiments on six strains of endemic typhus show that they lose their virulence more quickly than do strains from cases of the epidemic form of the disease.

At least two of the observations made in the preceding studies, i.e., (1) the fact that the organism is a bacillus, (2) and that it occurs in relatively small numbers in the blood, have a considerable bearing on the question of louse transmission. The presence of the few bacilli must render it relatively difficult for the lice to become infected, and this has been found by experiment. Lice only become infective five to six days after the first feeding, and the most probable explanation is one originally suggested by Wilder, that during this period the bacilli in the louse are undergoing an increase in numbers and virulence.

The authors also state that monkeys which have been exposed to the bites of infected lice may subsequently develop an immunity without having had fever or any other sign of the disease. This corresponds with the two cases mentioned before of individuals who developed agglutinins and complement-fixing antibodies after having been in contact with typhus cases without actually contracting the disease themselves.

From these experiments the authors have come to the following conclusions:—

(1) That a bacillus, identical with that recovered from patients with typhus, can also be isolated from the blood of animals in which the disease has been reproduced by the inoculation of typhus blood. In such animals the frequency of the bacillus in the blood is directly proportional to the severity of the disease. It is greatest at the height of the pyrexial period.

(2) That it is possible to produce the disease with the bacillus isolated from cases of typhus.

(3) That typhus blood is only infective if it contains a sufficient number of these bacilli. H. G. G.

JOURNAL

OF THE

ROYAL ARMY MEDICAL CORPS.

Corps News.

JUNE, JULY, AND AUGUST, 1915.

Q 180/34 No. L.C. 74/3.

General Headquarters,
Mediterranean Expeditionary Force,
August 28, 1915.

To I. G. C.

The Commander-in-Chief desires me to express to you his satisfaction with the way in which the wounded and sick have been evacuated from the Peninsula during the operations since August 7. So far as he can judge the business of evacuation has passed off practically without a hitch, and in his opinion it reflects great credit on all concerned.

Will you please express to all concerned his appreciation of the way they have worked, and of the success of their endeavours.

(Signed), G. F. ELLISON, Major-General, D.Q.M.G., M.E.F.

To D. D. M. S.

In forwarding above for your information and for communication to your staff, to Lieutenant-Colonel Garner, R.A.M.C., and to Medical Officers commanding hospitals, hospital ships, and temporary hospital ships, I desire to add my personal thanks for the devotion to duty, energy, and resourcefulness which enabled this satisfactory result to be attained.

(Signed), E. A. ALTHAM, Lieutenant-General, I.G.C., M.E.F.

Mudros, August 31, 1915.

War Office,
August 25, 1915.

His Majesty the King has been graciously pleased to approve of the appointment of the undermentioned officer to be a Companion of the Distinguished Service Order, in recognition of his gallantry and distinguished service in the Field:—

CAPTAIN STANLEY ALWYN SMITH, No. 3 FIELD AMBULANCE, CANADIAN ARMY MEDICAL CORPS.

For conspicuous gallantry and devotion to duty at Festubert on the night of May 20, 1915.

Captain Smith, with a party of eight men, went out voluntarily to remove the wounded from an orchard whilst under heavy fire, and eventually succeeded in bringing all into safety. Four of the eight men of the rescue party were wounded, and two of these have since died.

MILITARY CROSS. *July* 28, 1915.

The following notification appeared in the Supplement to the *London Gazette* of June 22, 1915 :—

His Majesty the King has been graciously pleased to approve of the undermentioned honours and rewards for distinguished service in the Field, with effect from June 3, 1915, inclusive :—

ROYAL ARMY MEDICAL CORPS.

AWARDED THE MILITARY CROSS.

No. 8268 Serjeant-Major G. B. Walker.

DISTINGUISHED CONDUCT MEDALS.

His Majesty the King has been graciously pleased to approve of the award of the Distinguished Conduct Medal to the undermentioned non-commissioned officers and men for acts of galantry and devotion to duty. (Second Supplement to the *London Gazette*, No. 29212, dated June 29, 1915.)

No. 12751 Serjeant T. B. Carter.

For most conspicuous and gallant conduct and devotion to duty at Houplines in tending the wounded under heavy fire, being often continuously on duty by day and night. This non-commissioned officer has previously displayed great gallantry in assisting the wounded under fire.

No. 7685 Private J. Cartwright.

For conspicuous devotion to duty since August, 1914, in tending the wounded frequently under fire. On February 25, 1915, he was reported for "most heroic" conduct under shell fire.

No. 587 Private A. W. Howitt.

For conspicuous gallantry and devotion to duty from December, 1914, to March, 1915, when in charge of an ambulance wagon. On more than one occasion he has volunteered for exceptionally dangerous duty, and during an action at Neuve Chapelle in October, 1914, was able to bring away two wagon loads after the troops had withdrawn from their positions.

No. 105 Serjeant F. Newman (now Staff Serjeant).

For conspicuous coolness and gallantry displayed during the periods November 16 to 20, 1914, and December 12, 1914, to March 31, 1915, when in charge of advanced dressing stations. In the presence of danger this non-commissioned officer has carried out his duties with great ability.

No. 11656 Serjeant H. J. V. Voisey.

For conspicuous gallantry throughout the campaign. He has displayed great coolness when collecting wounded at night and has shown excellent initiative on many occasions under fire. Serjeant Voisey performed very good work at the dressing station at Vailly in September, 1914, under very trying circumstances.

The Director General in recognition of the credit which these gallant actions reflect on the individuals concerned, and on the Corps, has directed that the Privates shall be promoted to the rank of Corporal as vacancies occur.

MENTIONED IN DESPATCHES.

The names of the following Warrant Officers, Non-commissioned Officers and men of the Royal Army Medical Corps, were mentioned in the despatch from the Field Marshal Commanding-in-Chief, British Forces in the Field, which was published in the *London Gazette*, No. 29200 of June 18, 1915.

Reg. No.	Rank and Name		Remarks
14050	Qmr.-Serjeant	Andrews, W.	
7438	Staff-Serjeant	Argent, W.	Acting Serjeant-Major.
7527	Private	Arnold, P. F.	
2217	,,	Avery, F.	
19106	,,	Bardwell, G. F.	
19812	Serjeant	Barron, F. P.	
10179	Private	Batchelor, P.	

Mentioned in Despatches—Continued.

No.	Rank and Name		Remarks
7174	Private	Benson, J. F.	
9115	,,	Blewitt, W. T.	
17964	Serjeant	Bowler, W.	Now Staff-Serjeant.
19665	,,	Boxall, H. G.	
7742	Private	Burdett, G.	
15027	Staff-Serjeant	Bush, W.	
18566	Corporal	Butler, H.	Now Serjeant.
7685	Private	Cartwright, J.	Now Corporal.
7247	,,	Chase, S. J.	
5190	Corporal	Clark, A.	
5018	,,	Cook, W. C.	
18222	Staff-Serjeant	Dady, A.	Now Qmr.-Serjeant.
20589	Private	Davis, W. J.	
19818	,,	Deakin, J. G.	
16115	Serjeant-Major	Dewberry, E. B. ..	
7695	Private	Emmerson, T.	
5145	,,	Evans, A.	
20732	,,	Floyd, H.	
14924	Staff-Serjeant	Forbes, J. G. A. ..	
8269	Serjeant-Major	Gibbs, G. A.	
10254	,,	Gillespie, A.	
14452	Corporal	Godfrey, F.	Now Serjeant.
20796	Private	Greenwood, H.	
284	Serjeant	Gregory, A. O.	
10831	,,	Gregory, H. W. G. ..	Acting Serjeant-Major.
16205	Qmr.-Serjeant	Gregson, T.	
2690	Private	Hamer, W. H.	
18443	Serjeant	Harris, G.	Now Staff-Serjeant.
9751	Private	Harrison, J.	
8633	,,	Hayne, E. W.	
18964	Serjeant	Herbert, G. W.	
1675	Private	Horrigan, P.	
18383	Serjeant	Hutchings, W.	,, ,,
17576	Staff-Serjeant	Ireson, J. R.	
17159	,,	Jones, C.	Acting Serjeant-Major.
2950	Private	Jones, J.	
10598	Qmr.-Serjeant	Knightley, P. G. ..	
17277	Private	Knowles, R. N.	
10221	Serjeant-Major	Larner, E.	
2484	Private	Last, W. A.	
1856	Serjeant	Leaney, A. F.	Now Staff-Serjeant.
12156	Private	Matchin, W.	
19863	Serjeant	Mattison, W. H. ..	
18330	,,	Mercer, J. N.	,, ,,
10073	Serjeant-Major	Merchant, W.	
3795	Corporal	Morrison, J.	
19509	Private	Mulley, H. F.	
18940	Qmr.-Serjeant	Musgrave, P. H. ..	
12506	Staff-Serjeant	O'Rourke, P. J.	
19296	Private	Owen, E. T. J.	
18453	Qmr.-Serjeant	Philbrook, F. A. ..	
19604	Serjeant	Pollock, R.	
19192	Staff-Serjeant	Poole, F.	
7536	Private	Pooley, A.	
113	Serjeant	Powell, J. D.	
1305	,,	Prince, H. M.	
15288	Qmr.-Serjeant	Prince, W. C.	
18158	Staff-Serjeant	Pursey, G. P.	Now Qmr.-Serjeant.
33169	Private	Reynolds, A. F.	

Mentioned in Despatches—Continued.

No.	Rank and Name		Remarks
10849	Qmr.-Serjeant	Richmond, C. E. T. ..	Now Serjeant-Major.
5600	Corporal	Roberts, R.	
14671	Staff-Serjeant	Robertson, W.	Now Qmr.-Serjeant.
754	Serjeant	Russell, H.	
11405	Staff-Serjeant	Scott, W.	
14290	Serjeant-Major	Scott-Badcock, W. H. ..	
1764	Serjeant	Selden, H. W.	
15483	Staff-Serjeant	Sharpe, E.	,, ,,
10711	Serjeant-Major	Sharpe, F. M.	
5648	Serjeant	Smith, E. F.	
11441	Serjeant-Major	Sprinks, H.	Now Quartermaster.
17568	,,	Steele, E.	
103	Staff-Serjeant	Steer, G. P.	
12779	,,	Stubbs, G.	Now Qmr.-Serjeant.
18772	Serjeant	Taylor, H. E.	
10200	Qmr.-Serjeant	Thomas, J. H.	Now Serjeant-Major.
19037	Serjeant	Thomas, W. B.	
6805	Private	Thorpe, B. P.	
17928	Staff-Serjeant	Toye, W. S.	Now Qmr.-Serjeant.
9467	,,	Tunn, C. J.	Acting Serjeant-Major.
8268	Serjeant-Major	Walker, G. B.	
14008	,,	Watt, D.	Now Quartermaster.
12302	Staff-Serjeant	Whyte, W.	
20852	Private	Wilkinson, H.	

His Imperial Majesty the Emperor of Russia has been graciously pleased to confer, with the approval of His Majesty the King, the undermentioned rewards for gallantry and distinguished service in the Field :—

THE ORDER OF ST. ANNE 4TH CLASS, INSCRIBED "FOR VALOUR IN WAR."

Lieutenant John Downie, M.B., Yorkshire Mounted Brigade Field Ambulance, Royal Army Medical Corps, Territorial Force.

CROSS OF THE ORDER OF ST. GEORGE 4TH CLASS.

No. 336 Serjeant Arthur William Brown, 81st Field Ambulance, Royal Army Medical Corps, Territorial Force.

No. 32713 Serjeant Tyler Morris Brown, No. 1, Canadian Field Ambulance.

No. 12751 Serjeant Thomas Baylis Carter, 19th Field Ambulance, Royal Army Medical Corps.

No. 35664 Serjeant Richard Rodney Davies, 14th Field Ambulance, Royal Army Medical Corps.

No. 11628 Serjeant Thomas Arthur Fullam, Royal Army Medical Corps.

No. 2484 Private William Arthur Last, 5th Cavalry Field Ambulance, Royal Army Medical Corps.

No. 972 Private Henry Charles Sell, 84th Field Ambulance, Royal Army Medical Corps, Territorial Force.

No. 7604 Private David Wolfe, No. 5 Field Ambulance, Royal Army Medical Corps.

MEDAL OF ST. GEORGE, 1ST CLASS.

No. 551 Serjeant Charles Ingram, 85th Field Ambulance, Royal Army Medical Corps, Territorial Force.

MEDAL OF ST. GEORGE 2ND CLASS.

No. 14077 Corporal Frank Malcolm Harris, Royal Army Medical Corps.

No. 18576 Staff-Serjeant William Lamkin, Royal Army Medical Corps.

MEDAL OF ST. GEORGE, 3RD CLASS.

No. 9939 Corporal Alexander Burns, Royal Army Medical Corps.

No. 7766 Private Walter Richard Fitch, Royal Army Medical Corps (attached 1st Battalion Hampshire Regiment).

Driver John Vicary Gibbs No. 1195, British Red Cross Society (attached No. 4 Ambulance Convoy).

No. 6805 Private Benjamin Patrick Thorpe, Royal Army Medical Corps.

No. 33191 Private Frank Turner, No. 2 Canadian Field Ambulance.

MEDAL OF ST. GEORGE, 4TH CLASS.

No. 7695 Private Thomas Emmerson, Royal Army Medical Corps (attached 2nd Battalion, King's Own Scottish Borderers).

No. 3311 Private William Hanson, No. 1 Field Ambulance, Royal Army Medical Corps.

No. 284 Private William Malone, St. John Ambulance Association (attached No. 4 Motor Ambulance Convoy).

No. 1249 Private Thomas Markes, 1st Cavalry Field Ambulance, Royal Army Medical Corps.

No. 3890 Sepoy (Ward Orderly) Sadardin, 59th Scinde Rifles (Frontier Force) (attached 112th Indian Field Ambulance).

No. 3177 Private Henry Stapleton, 3rd Field Ambulance, Royal Army Medical Corps, Territorial Force.

No. 182 Private Philip Mark Stephens, 86th Field Ambulance, Royal Army Medical Corps, Territorial Force.

No. 33470 Private Charles Barnard Tomkins, No. 3 Canadian Field Ambulance.

No. 20852 Private Harry Wilkinson, 18th Field Ambulance, Royal Army Medical Corps.

No. 2304 Private Albert E. Wright, 81st Field Ambulance, Royal Army Medical Corps, Territorial Force.

There are no restrictions as to the occasions on which these decorations may be worn. No individual applications for permission to wear them need, therefore, be submitted.

ARMY MEDICAL SERVICE.

Lieutenant-Colonel (temporary Colonel) Richard R. Sleman, M.D., Royal Army Medical Corps (Territorial Force); to be an Assistant Director of Medical Services, and to retain his temporary rank whilst so employed, dated August 5, 1915.

Colonel Robert J. Geddes, D.S.O., is retained on the Active List, and to be supernumerary, dated August 13, 1915.

Andrew Fullerton, F.R.C.S.I., to be temporary Colonel, dated September 1, 1915.

ROYAL ARMY MEDICAL CORPS.

Temporary Lieutenant (temporary Honorary Lieutenant-Colonel) Arthur S. Woodwark, M.D., to be temporary Lieutenant-Colonel, dated August 12, 1915.

The undermentioned to be temporary Majors:—

Dated June 12, 1915.—William Forsyth Jones to be temporary Major whilst employed with the Brook War Hospital.

Dated August 12, 1915.—John Harley Brooks, M.D., to be temporary Major whilst serving with the Mile End War Hospital.

Dated August 14, 1915.—Temporary Lieutenant Robert C. Brown, M.D., whilst employed in charge of the Springburn and Woodside Hospital, Glasgow.

Dated August 18, 1915.—Temporary Lieutenant Thomas W. Buckley, M.D., to be temporary Honorary Major whilst serving with the Princess Christian Hospital.

Dated August 21, 1915.—Temporary Captain George Stoker.

The undermentioned to be temporary Majors:—

Dated September 1, 1915.—Hamilton Clelland Marr, M.D.; Francis Martin Rouse Walshe, M.D.

The date of appointment of Hamilton Irving, M.B., F.R.C.S., as temporary Major, is September 1, 1915, and not as stated in the *Gazette* of July 28, 1915.

Ernest Frederick Eliot, F.R.C.S.Edin., to be temporary Major, dated April 11, 1915. (Substituted for the notification which appeared in the *Gazette* of June 23, 1915.)

The undermentioned temporary Lieutenants to be temporary Captains:—

Dated July 15, 1915.—Henry William Martindale Kendall, late Captain, Army Medical Staff.

Dated August 5, 1915.—Ernest E. S. J. Galbraith; William T. Hessel, M.B.; Arthur T. Todd, M.B.; Frederick W. Robinson, M.D., F.R.C.S.; Frank Whitby, M.B.

Dated August 6, 1915.—Richard B. Lilly.

Dated August 7, 1915.—Harold A. Douglas, M.B.; Malcolm Donaldson, M.B., F.R.C.S.; Arthur J. Eagleton, M.B.; William Kelsey-Fry; Horace W. Hay; Charles S. P. Hamilton; William M. Howells, M.B.; George D. Jameson; William Allan, M.B.; Hamish M. Anderson, M.B.; Kenneth B. Aikman; Henry W. Batchelor; St. John D. Buxton; Howard A. Bell; Sidney A. Boyd, F.R.C.S.; John A. Cowan, M.B.; Louis Lazarus; Harold Y. Mansfield, M.B.; Reginald K. MacGregor; Alexander F. Potter; Duncan W. Pailthorpe; Henry D. Robb, M.B. William H. D. Smith, M.B.; Ernest Scott, M.B.; John Fraser Taylor, M.B.; Herbert A. Watermeyer; Martyn H. Watney, M.B.; Charles S. E. Wright, M.B.; Owen L. V. de Wesselow, M.B.; Arthur J. Waugh; Ivan S. Wilson, M.D., F.R.C.S.

Dated August 8, 1915.—William H. Lister; Charles W. B. Littlejohn, M.B.; Rupert S. Scott, Vernon C. W. Vickers; Henry B. Owens; Percy P. Butler.

Dated August 9, 1915.—Francis Henderson, M.B; William E. Hallinan; Eric L. MacKenzie, M.B.; Hyacinth B. Morgan, M.B.; Herbert O'Callaghan, M.B.; Robert K. Sutherland, M.B.; William G. Waugh, M.D.; Lionel D. Woods; Thomas L. Fraser, M.B.; Stanley Arnott, M.B.; Edward Billing.

Dated August 10, 1915.—Frederick C. Davies, M.B.; Wilberforce V. Eaves, M.D.; Walter Groome, M.B.; Arthur S. Glynn, M.B.; John Greene; William G. Goudie, M.B.; John B. Haycraft, M.D.; William O. Halpin, M.D.; George M. W. Hodges, M.B.; Cecil B. Hogg, M.B.; Claude C. Harrison, M.B.; Ronald Hodson, M.B.; John L. Jackson, M.B.; Geoffrey T. Loughborough; Alan Mann, M.B.; Colin Mackenzie, F.R.C.S.; Hugh W. Moir, M.B.; Findlay Murchie, M.B.; Hugh J. Orr-Ewing, M.B.; Francis W. O'Connor; James E. H. Roberts, M.B., F.R.C.S.; Wilfred A. Russell, M.B.; Edmund L. Reid, M.B., F.R.C.S.Edin.; William J. Stewart, M.B.; Maitland Scott; Cyril Sherris, M.B.; Ronald Silcock; Reginald S. S. Statham, M.D.; Lionel H. Y. Stephen; James S. Stewart, M.B.; Thomas V. Somerville; Gerard A. Smythe, M.B.; Robert S. Snowie, M.B.; Vincent Townrow, M.B., F.R.C.S.; Cyril E. Thwaites; Hugh D. Willis, M.B.; Frederick E. S. Willis; Eric Wordley, M.B.; Charles H. S. Webb, M.B., F.R.C.S.; Donald McD. Wilson, M.B.; Philip R. Woodhouse, M.B.; Charles Weller, F.R.C.S.; Humphrey B. Wilson, M.D.; Basil W. Armstrong; William R. Addis, M.B.; Norman Briggs; Lancelot G. Bourdillon; Gavin S. Brown, M.B.; John B. Burgess, M.D.; Arthur E. Bullock; Monamy A. C. Buckell, M.B.; Bertram F. Bartlett; Charles N. Binney, M.B.; Cryil J. W. Clayton; George M. Campbell, M.B.; David H. Clarke, M.B.

Dated August 11, 1915.—Walter S. Danks, M.D.; William F. Evans, M.D.; Wilfred F. Hawkins, M.B.; Leonard L. Hadley, M.B.; William H. Johnston; Herbert H. P. Morton; Henry Moore; John Morris; Victor E. Negus; Duncan J. McRae, M.B.; Norman P. Pritchard; William A. Ryan, M.B.; Harold B. G. Russell; Nicol McN. Rankin, M.B.; Robert L. Roe, M.B.; Adrian D. Stokes, M.D., F.R.C.S.I.; Ralph L. Scott, M.B., F.R.C.S.Edin.; Francis R. Thornton, M.B.; Henry E. Wiltshire; Harold E. McM. Wall; John B. Young, M.B.; Ralph D. O'Leary, M.B.; Edward W. Carrington, M.B.; Alexander B. Cheves, M.B.

Dated August 12, 1915.—Cedric L. Dold, M.B.; Henry de C. Dillon, M.B.; James H. Dible, M.B.; Charles H. Evans, F.R.C.S.Edin; Joseph H. Elliott, M.D.; John McI. Falkiner, F.R.C.S.I.; James R. C. Greenlees, D.S.O., M.D.; Joseph G. Greenfield, M.B.; Thomas H. Holroyd, M.B.; George W. Kendall, M.D.; Thomas M. Low, M.B.; George A. Lilly; Clement R. Macleod, M.B.; Stuart A. O. Mackenzie, F.R.C.S.Edin.; Henry F. Mullan; Sidney H. Nathan, M.D.; Montgomery P. Paton, M.B.; Charles M. Row; James H. Ritchie, M.B.; William L. Scott, M.B.; Herbert W. Smith, M.B.; Frederick J. Thorne, M.B.; Russell E. Walker, M.B.; Frederick J. Whitelaw, M.B.; Edward A. Aldridge; Henry J. Burke; Cyril A. Bernard.

Dated August 13, 1915.—Kenneth B. Dickson; Charles H. Denham, M.B.; Alexander R. Esler; William B. Gordon; Oliver K. Hartridge, M.B.; Frederick T. Hill; James E. T. Jones; James D. Jones; Trevor A. Lawder, M.B.; Thomas Martin, M.B.; Hugh J. More, M.D., F.R.C.S.; Samuel W. McLellan, M.D.; Frederick L. Napier, M.B.; George Rickman; Charles M. Rout, M.B.; David H. Russell, M.D., F.R.C.S.Edin.; Granville D. Robertson; Robert B. Rutherford, M.B.; John Sainsbury, M.B.; George W. Smith, M.B.; Arthur F. S. Sladden, M.D.; Humphrey M. Hart-Smith, M.B.; Herbert Walker; Spencer L. Walker, M.B.

Dated August 14, 1915.—William Duguid, M.B.; George Elkington, M.B.; George

D. Ferguson, M.B.; Gilbert L. K. Finlay, M.B.; Geoffrey P. Humphery, M.B.; Robert H. C. Lyons, M.B.; George R. D. McGeagh; Edward J. Nangle; Edward O'Connor, M.B.; Arthur S. Plant; Hubert A. Pallant; John W. Pell; John F. G. Richards, M.B.; Carlyle Aldis, M.D.; Robert M. Alcorn; Cecil Bluett; Aleck W. Bourne, M.B., F.R.C.S.; Gurth S. Blandy, M.D.; John D. Carroll, M.B.

Dated August 15, 1915.—Robert V. Dolbey, F.R.C.S.; Richard E. Gibson, M.B.; Cameron R. Gibson, M.B.; Charles E. Hibbard; John H. Hood, M.B.; Frederick M. S. Hulke; Thomson Henderson, M.B.; Henry J. R. Jones; Basil T. Lang, F.R.C.S.; Robert E. Lee, M.B.; John B. Lowe, M.B.; George C. M. M'Gonigle, M.D.; Leslie Meakin; John B. Matthews; Philip W. Maclagan, M.B.; Henry F. Marris, M.B.; John A. MacLeod, M.B.; William B. Purchase; Andrew E. S. Pringle-Pattison, M.B.; Henry E. Peake, M.B.; Joseph A. Quin, M.B.; Alexander A. Rees, M.D.; Edwin C. Rayner; Harold Sheldon, M.B.; Hugh L. Sells, M.B.; Lawrence L. Satow; Richard W. Smith, M.B.; James L. Stewart, M.B.; Victor F. Scotbill, M.B.; Douglas W. Smith, M.B., F.R.C.S; Arthur Tilbury; Oscar R. L. Wilson, M.B.; Alfred S. Wakeley; Frederick B. Winfield; Robert J. B. Madden, M.B.; Reginald H. Jones, M.B.; William D. Arthur; James N. Armstrong, M.B.; George N. Braham, F.R.C.S.Edin.; Hamish D. F. Brand, M.B.; Arthur Joseph Blake.

Dated August 16, 1915.—Philip Ferguson, M.B., F.R.C.S.; Edward G. Foley; Carleton Y. Ford, M.D.; James F. Fairley, M.B., F.R.C.S.; Ernest H. Griffin, M.D.; Thomas Hardy, M. B.; Theodore H. Just, M.B.; Geoffrey L. Keynes; George J. W. Keigwin; Joseph P. Little; Edmund H. Moore, D.S.O., M.B.; Adolph R. N. MacGillycuddy; John H. Meers; Kenneth P. Mackenzie, M.B.; Edward Thomas C. Milligan, M.D.; Sydney F. McDonald, M.D.; Ernest S. Miller, M.D.; Hector Mortimer, M.B.; Robert M. Miller; Charles J. O'Reilly, M.D.; Maurice N. Perrin; Thomas E. Parrer, M.B.; Charles P. Porter; William E. Reid, M.B.; Horatio F. N. Scott, M.D.; William V. T. Styles; Alexander W. Uloth; Bernard H. Well, M.D.; Thomas S. Allen; George G. Anderson, M.B.; Henry Cordner, M.B.; Francis H. Cleveland; William L. Cassells, M.B.; Frank D. Cairns, M.B.

Dated August 17, 1915.—John M. Gillespie, M.B.; George W. L. Kirk, M.B.; David C. Monro, M.B.; Herbert F. W. Adams, M.B.; Frederick C. Atkinson-Fleming, M.B.; Alexander Baldie, M.B.

Dated August 18, 1915.—William Foot, M.B.; Alexander T. I. MacDonald, M.D.; Frederick C. K. Austin, M.B.; Allan L. Christie, M.B.

Dated August 19, 1915.—John C. Hallinan; Ivan C. Maclean, M.D.

Dated August 20, 1915.—Arthur W. Dennis, M.B.; Byron L. Hutchence; Arthur D. Haydon; Arthur G. Maitland-Jones; John W. Linnell, M.D.; Robert R. Archibald, M.B.; Ian M. Brown.

Dated August 22, 1915.—Walter Dawson, M.B.; Cosmo W. Fowler, M.B.; Rudolf W. Galloway, M.B.; Harry L. S. Griffiths; Robert H. Hodges; Ronald N. Hunter; Charles H. Hart, M.B.; William Henderson, M.B.; Francis H. Moxon, M.B.; John M. McLaggan, M.B.; Francis L. Nash-Wortham, F.R.C.S.Edin.; John Proctor, M.B.; William D. Reid, M.B.; Andrew Topping, M.B.; Edward H. Udall; Gerald N. B. Sebastian; Leslie Adamson, M.D.; Galvin A. E. Argo, M.B.; Edward W. Archer, M.B.; Robert H. Alexander, M.B.; Mark B. Baines, M.D.; John S. Buchanan, M.B.; David D. Craig, M.B.; Spencer S. Crosse.

Dated August 28, 1915.—John Lewis Maitland Govan.

Dated September 8, 1915.—Temporary Lieutenant Francis Charlesworth, M.B.

The undermentioned temporary Lieutenants relinquish their commissions:—

Dated August 13, 1915.—Norman F. Hallows, M.B.; George H. Varley, M.B.; Rex Stansfeld; Alan D. Anderson; Aubrey D. Vernon-Taylor; Algernon C. S. Smith; David L. Lewis; William A. Stewart; Herbert W. Cooke.

Dated August 14, 1915.—Horace F. W. Warden; John Hewat, M.D

Dated August 15, 1915.—Dumaresq Le Bas; David Morrow; Henry D. Gasteen; Raymond B. Taylor, M.B.; James J. Woodburn, M.B.; Andrew D. Carberry, F.R.C.S.I.; Hugh P. Costobadie, F.R.C.S.Edin.; Sidney T. Davies; Harold B. Whitehouse, F.R.C.S.; Edward E. Steele, M.D.; Arthur N. Hodges, M.B.

Dated August 16, 1915.—Richard D. Passey, M.B.; Henry L. Martyn, M.B., F.R.C.S., Harold E. Battle.

Dated August 17, 1915.—James A. Raeburn, M.D.: Alister F. Cowan, M.B.; Arthur Turnbull, M.B.

Dated August 22, 1915.—Gideon R. E. Colquhoun; Durie A. Chamberlain; Thomas Gilchrist, M.B.; Douglas McAlpine, M.B.; Issachar R. Smith, M.B.; Thomas S. Stafford; Michael J. Cronin, M.B.; Cresswell L. Pattison, M.B.; John M. Wilson.

The undermentioned to be temporary Lieutenants:—

Dated July 3, 1915.—Charles Richard Whittaker, F.R.C.S.Edin.

Dated July 10, 1915.—James Mitchell Whyte, M.B.; Alexander Frew, M.D.; Francis C. Drew, M.B.; John Michael Verster, M.B.: Ernest France Watson, M.B.

Dated July 25, 1915.—Richard William Ely Roe.

Dated July 16, 1915.—Lieutenant Norman Burke Taylor, M.B., F.R.C.S.Edin., Canadian Army Medical Corps.

Dated July 24, 1915.—James Mary Joseph Allen Levines; Ernest Louis Marsh, M.B.

Dated July 25, 1915.—Charles Gerald Harmer, M.D.

Dated July 28, 1915.—Frederick Arthur Martin Flegg; Frederick Wyndham Chamberlain; Melville Krolik, M.D.; Owen Douglas Price, M.B.; John Pritchard; Thomas Lindsay Clark, M.B.; William Noel Child; Donald Ewart Morley, M.B.; John Rodger, M.B.; Charles Augustus Eamonson Ring, F.R.C.S.Edin.; Sydney Victor Shrimpton; Thomas Morton Johnston Stewart, M.B.; William Graham Scott; Frederick Naylor Stewart, M.D.; Harold Hope Scott; William Turner, M.B.; Maurice Exell Willcock, M.P.; John Hemphill Rutter, M.B.

Dated July 29, 1915.—George William Elder, M.B.; John Pearce Whetter, M.B.; Murdo McKenzie McRae, M.B.; Austin Harvey Huycke, M.D.

Dated July 30, 1915.—Walter Weir Galbraith, M.B.; Frederick Alexander Anderson, M.D.; Reginald Anson Mansell, M.B.; James MacCormac Caldwell Johnston, M.B.; Robert Stewart Ross, M.B.; Gerald Struan Marshall.

Dated July 31, 1915.—John James Mackintosh, M.B.; Diederick Johannes Dauth, M.B.; James Frederick Matheson, M.B.; George Elphinstone Keith, M.B.; Ralph Cox, M.B.; Edward Melville Bruce Payne; John Carr Robertson, M.B.; Charles Lyle Sproule; Charles Kidd, M.B.; Joseph Dunlop, M.B.

Dated August 2, 1915.—Ralph Boutwood.

Dated August 3, 1915.—Arthur William Johnson.

Dated August 4, 1915.—Everard William Lewen Sharp to be temporary Honorary Lieutenant.

Dated August 8, 1915.—McWilliams Henry, F.R.C.S.Edin.

Dated August 9, 1915.—George Stewart Clark, M.D.; William Allan Young, M.B.; Francis George Cross, F.R.C.S.; John Reid, M.D.; Charles Dickson, M.D.; Harry Stokes; William John Gibson; John Walker; Edward Mountjoy Pearse; Robert Wilson, M.B.; Hamilton Brown Lord Henderson, M.B.; George Charles Gaynor, M.B.; Josiah Rowland Benjamin Dobson, M.B.; Thomas Herrick Sarsfield; Douglas James Glen, M.B.; George Joughin, M.B.

Dated August 10, 1915.—Lionel John Lock; Philip Rutherford Boswell; Alexander Urquhart Webster, M.B.; Charles Renfric Chichester, M.B.; Frederick Reginald Sturridge; Joseph Paterson Lusk, M.B.; Harold Reginald Wessen Husbands; Blacker Castles Powell, M.B.; William Rickard Lloyd Waters, M.B.; Herdman Porter, M.B.; Frank Percival Montgomery, M.B.; Robert Boyd Robson, M.B.; Lorimor Gifford Nash.

Dated August 11, 1915.—Robert Hugh Russell McKean; John Inman Langley; Noel Archibald Scott; John Vincent Bates.

Dated August 12, 1915.—Bertram Chiene Letts, M.B.; Joseph Vincent Duffy; James Patrick O'Hea, M.B., F.R.C.S.; Arthur Walton Rowe, M.B.; James Alexander Cowie, M.D. F.R.C.S.Edin.; Frederick Vaudry Cant; Russell Walbancke Hodgson-Jones; James Hepburn, M.B.; Harold Pringle, M.D., F.R.C.S.I.; Charles William Sanderson Davies-Jones, M.B.; Robert Arthur Welsford Proctor; William John Hogg.

Dated August 13, 1915.—Edward Leonard Taylor; James L. Wilson, M.B.; Charles James Stanley, M.B.

Dated August 14, 1915.—Harry Seymour Laird; Eric Biddle; Vernon Edmund Lloyd; Robert Ferguson Copland, M.B.; Carmel Samut, M.D.; Joseph Morgan Richey, M.B.; Stanley Ernest York Elliott; Arthur Willatt; John Tolmie MacKenzie; William Waddell, M.D.; James Lawson Russell, M.B.; Arthur George Troup, M.D.; Robert Marshall Hume, M.B.; Charles Patrick Kelly, M.B.; Kenneth Noel Purkis; William Irving, M.D.; Edmund Benjamin Jones, F.R.C.S.; Joseph Walker, M.D.; Patrick George Milne, M.B.; William Campbell, M.B.

Dated August 15, 1915.—Arthur George Mossop.

Dated August 16, 1915.—John Colley Pounden Beatty, M.B.; Thomas Jackson Gilmore, M.B.; Arthur Frederick Cole; Frederick Otto Stohr, M.D.; Maurice Smith Bryce, M.B.; Lionel Eugene Sutcliffe, M.B.; Dennis Cregan McCabe-Dallas; Morris John Theodore Wallis; William Thomas Ingestre Abell.

Dated August 17, 1915.—Herbert Wales, M.B.; William Forsyth, M.B.

Dated August 18, 1915.—Fritz Salo Eschwege; John Beattie McFarland; Charles Reginald Hoskyn, M.D.; William George Theaker Story, M.B.; Richard Athlestane Parker Hill, M.D.; Robert Henry Wilson, M.B.; Terence Patrick McQuaid, M.B.; John Campbell, M.B.; Colin Campbell Blair Gilmour, M.B.; Andrew Gibson, M.B., F.R.C.S.Edin.; George Francis Palmer Heathcote, M.B.; James Alphonsus Conway, M.D.; William Cullen, M.D.; David Macquorn-Rankine Crighton, M.B.; Thomas Scott Brodie, M.B.; Patrick Joseph Lane, M.B.; Ewan Gordon Cameron; Robert Fenwick Linton, M.B.; Charles Carrick Brewis.

Temporary Lieutenant Harold McAllum, M.B., relinquishes his commission, dated October 17, 1914.

Lieutenant Ian Macdonald, M.D., F.R.C.S.Edin., relinquishes his temporary commission on account of ill-health, dated July 29, 1915. (Substituted for the notification which appeared in the *Gazette* of May 22, 1915.)

The date on which the undermentioned temporary Lieutenants relinquish their commissions is August 7, 1915, and not as stated in the *Gazette* of August 18, 1915:—

Maitland Radford, M.B.; Gilbert C. Chubb, M.D., F.R.C.S.; Robert G. Brown; Alma P. Ford; Frederick G. Chandler, M.B.; George C. Metcalfe; Arthur G. H. Lovell, M.D., F.R.C.S.

The undermentioned temporary Lieutenants relinquish their commissions:—

Dated August 9, 1915.—Henry Robinson, M.D.; Arthur E. Brown; Bernard V. Dunne, M.B.

Dated August 10, 1915.—John S. Avery, M.B.; John Dotto; Montague S. Woolf; John Whigham, M.B.; Philip W. James, M.D.; Cornelius Molan; Robert J. Harley-Mason.

Dated August 11, 1915.—Charles S. Atkin; Cecil K. Attlee; Edward N. Graham, F.R.C.S.; Neville C. Wallis; John A. West.

Dated August 12, 1915.—Thomas A. Jones, M.D.; Andrew B. Lindsay, M.B.; Charles W. Forsyth, M.B.; William A. Wilson-Smith, M.D.; Henry G. Greaves, M.B.

The notification regarding Ralph Boutwood and Arthur William Johnson, which appeared in the *Gazette* of August 19, 1915, is cancelled.

Temporary Lieutenant Frederick G. Thomson, M.B., relinquishes his commission on account of ill-health, dated September 1, 1915.

Temporary Lieutenant William R. A. Coates relinquishes his commission on account of ill-health, dated September 4, 1915.

The undermentioned to be temporary Quartermasters, with the honorary rank of Lieutenant:—

Dated August 16, 1915.—Alfred Jackson.

Dated August 20, 1915.—John Suffield Whestone.

Dated August 22, 1915.—John Goodwin.

Dated August 25, 1915.—Francis George Payne.

Dated August 28, 1915.—John Thomas Cooke; Herbert Hargreaves Donnelly; William Frederick Grocott; William John Cyril Merryman; Edwin Turner Grundy.

Dated August 30, 1915.—George Edward Barney; John Watson.

Dated August 31, 1915.—Archibald James Andrews.

Dated May 5, 1915.—Quartermaster and Honorary Captain Edwin Houghton, retired pay, Royal Army Medical Corps, to be honorary Major.

WARRANT OFFICERS, NON-COMMISSIONED OFFICERS, AND MEN.

The following promotions to complete War Establishment will take effect from the dates specified:—

To be Quartermaster-Serjeants.

No.	Rank and Name		Date	Remarks
10511	Staff-Serjt.	Morgan, F.	1.7.15	
13035	,,	Chandler, G. V. ..	7.7.15	
17358	,,	Ennor, C.	,,	
12709	,,	Ford, H. J.	15.7.15	
17091	,,	Moore, J.	19.7.15	
19320	,,	Ritchie, H. A. ..	24.7.15	

To be Staff Serjeants.

No.	Rank and Name	Date	Remarks
12547	Serjeant Triggs, A.	1.7.15	
19032	,, Cooke, J.	,,	
19282	,, Golden, H.	7.7.15	
1843	,, Mack, C. A.	,,	
1905	,, Eves, J. G.	,,	
19747	,, Hyde, C. H.	,,	
1620	,, Pegg, A. E.	15.7.15	
18898	,, Green, G. H.	,,	
19086	,, Wain, A.	19.7.15	
19193	,, Stebbings, W. M. ..	,,	
18604	,, Tindall, W.	24.7.15	
18982	,, Newman, A.	,,	

To be Serjeants.

5108	Corporal	Westwood, C.	20.1.15	As being in possession of A.F.C. 344. With seniority next below No. 5023 Serjeant R. Grist.
19128	,,	Burr, W. G.	9.5.15	As being in possession of A.F.C. 344. With seniority next below No. 12650 Serjeant E. Wyke.
11894	,,	Snow, R.	,,	
19698	,,	Wass, M.	13.5.15	As being in possession of A.F.C. 344. With seniority next below No. 15289 Serjeant H. R. M. Rodman.
1556	,,	Calvert, N. B. ..	1.6.15	As being in possession of A.F.C. 344. With seniority next below No. 12344 Serjeant F. H. Lucas.
11414	Lance-Serjt.	Wade, H. J.	28.6.15	
1430	Corporal	Perkins, W. T. ..	29.6.15	Special as clerk.
1490	,,	Lansdowne, E. W. ..	1.7.15	As being in possession of A.F.C. 344.
4962	,,	Matheson, J.	,,	Special as clerk.
17215	,,	Lilley, W. A.	7.7.15	—
18714	,,	Knight, J.	,,	—
16979	,,	Egan, R. V. V. ..	,,	—
17894	,,	Syrett, G. R.	,,	
19135	,,	Quelch, W. H. ..	15.7.15	As being in possession of A.F.C. 344.
18243	,,	Vincent, W.	,,	
18506	,,	Cray, R. W.	19.7.15	—
18524	,,	Smitherman, T. H. ..	,,	—
16155	,,	Clarke, G.	24.7.15	—
18689	,,	Appleton, C.	,,	—

To be Corporals.

No.	Rank and Name		Date	Remarks
2281	Private	Edington, T.	28.6.15	
5843	,,	Fraser, J.	,,	
7744	,,	Cronin, J. L.	,,	
5844	,,	Dilks, M.	,,	
4635	,,	Pawley, G. E. ..	,,	
5106	,,	Clarke, A. E.	,,	
5263	,,	Romney, T. A. ..	,,	
5371	,,	Dunne, D.	,,	
5386	,,	Gray, F. A.	,,	
5482	,,	Laird, A. G.	,,	
6439	,,	L'Arminie, G. ..	,,	
6479	,,	Chifney, T. G. ..	,,	
20271	,,	Morris, J.	,,	
20467	,,	Drew, A. J.	,,	
20601	,,	Naude, A. E.	,,	
7685	,,	Cartwright, J. ..	29.6.15	In accordance with Corps Order No. 58 of this date.
587	,,	Howitt, A. W. ..	30.6.15	
5641	,,	Brown, T.	1.7.15	
5630	,,	Ely, T. O.	,,	
12346	,,	McReavie, D.	4.7.15	
5808	,,	Maxwell, M. M. D. ..	7.7.15	
5884	,,	Jay, E. B.	,,	
5905	,,	Elliott, W.	,,	
5952	,,	Rendell, C. E. ..	,,	
15724	,,	Bush, C. H.	15.7.15	
6017	,,	Moody, H.	,,	
12318	,,	Dart, F. C.	19.7.15	
2904	,,	Mossop, G.	,,	
2652	,,	White, F. W.	24.7.15	
2843	,,	Brown, A.	,,	
3886	,,	Palmer, F. E. ..	,,	

These promotions are subject to the conditions laid down in paragraph 35 Standing Orders, Royal Army Medical Corps, 1914.

AWARD OF ARMY FORM "C. 344."

The undermentioned have been awarded Army Form C. 344 on completion of three years' training in accordance with Paragraph 330 Standing Orders on the dates specified :—

No.	Rank and Name		Date	Remarks
5108	Corporal	Westwood, C.	13.1.15	
1569	Serjeant	Farmer, G. L. ..	28.6.15	
12819	Staff-Serjt.	Riches, W. H. ..	21.7.15	

NURSING SECTION.

The following appointments to the Nursing Section of the Corps will take effect from the dates specified:—

No.	Rank and Name	Date	No.	Rank and Name	Date
9596	Private Cooper, F. ..	14.6.15	5874	Private Cripps, A. W.	28.6.15
4499	,, Browning, S...	20.6.15	2718	,, Gilburd, E. ..	,,
5985	,, Burke, P. ..	,,	19165	,, Faith, A. E. ..	,,
4486	,, Campion, B. R.	,,	2928	,, McKinley, T.	,,
7541	,, Midgley, C. ..	,,	868	,, Cobie, H. T. ..	3.7.15
1570	,, Gorton, C. ..	,,	18499	,, Haveron, J. ..	,,
2458	,, Delaney, C. ..	,,	1257	,, Fitzpatrick, D. C.	,,
4464	,, Sewell, F. J...	,,			
392	,, Sparks, J. ..	21.6.15	2351	,, Brown, M. ..	,,
10718	,, Stone, R. F. ..	,,	5815	,, Hearnden, W.	,,
10642	,, Allan, H. ..	,,	5787	,, Griffiths, L. J.	3.7.15
8589	,, Arnold, C. ..	,,	7797	,, Firth, G. W...	,,
8945	,, Bass, A. ..	,,	10627	,, Porter, H. ..	5.7.15
8942	,, Green, G. H...	,,	400	,, Gallagher, P.	,,
10321	,, Kent, C. W. ..	,,	7529	,, Capel, J. W...	6.7.15
8898	,, Patterson, D.	,,	6971	,, Collier, G. E.	,,
10226	,, Tozer, W. ..	,,	12641	,, Hurst, A. H...	,,
8940	,, Vallender, J. J.	,,	7799	,, McCord, S. ..	,,
3397	,, Neary, M. ..	22.6.15	5809	,, Pitt, P. ..	,,
4476	,, Hewitt, G. ..	,,	20314	,, Sheen, E. ..	,,
10169	,, Morgan, H. ..	23.6.15	4723	,, Samson, W. ..	,,
10533	,, Sutherland, G.	,,	6399	,, Taylor, B. L...	,,
2761	,, Taylor, R. J...	,,	502	,, Thornhill, R..	,,
1188	,, Doyle, M. ..	24.6.15	3508	,, Armstrong, A.	,,
19116	,, Dodgson, T. .. (re-appointed)	,,	16374	,, Barney, E. H.	,,
			20148	,, Bell, E. J. S...	,,
5302	,, Hayter, J. W.	,,	6537	,, Bedford, F. A.	,,
3109	,, Rice, S. ..	,,	20407	,, Bryan, T. W.	,,
6114	,, Love, G. G. ..	,,	20462	,, Chadwick, A...	,,
3491	,, Marston, W...	,,	17715	,, Claydon, G. ..	,,
2553	,, McCullagh, H.	,,	209	,, Croxford, C. ..	,,
2555	,, Molloy, T. ..	,,		(re-appointed)	
28	,, Bracken, J. ..	,,	17782	,, Curry, H. ..	,,
500	,, Majury, J. ..	,,	20610	,, Elliott, W. A.	,,
1702	,, Ranson, A. E.	,,	4883	,, Long, J. W. ..	,,
3232	,, Smyth, J. ..	,,	6129	,, McDermott, J.	,,
10679	,, Upfold, L. A.	,,	7864	,, Merrill, C. C.	,,
480	,, Bell, J. ..	25.6.15	2845	,, Moore, F. ..	,,
3542	,, Brammeld, H.	,,	5519	,, Nicolle, W. ..	,,
3541	,, Cameron, J. ..	,,	7754	,, Reast, A. W.	,,
3579	,, Deans, J. ..	,,	10800	,, Simpson, H...	,,
3522	,, Doyle, P. ..	,,	4633	,, Sowden, C. ..	,,
4121	,, Doherty, G. ..	,,	136	,, Wood, C. H...	,,
3521	,, Kennedy, M...	,,	7841	,, Hendry, J. ..	11.7.15
2570	,, Kerwin, P. ..	,,	861	,, Matheson, A..	12.7.15
71	,, Lane, C. H. ..	,,	7433	,, Berryman, H.T.	14.7.15
6139	,, Mahoney, J...	,,	10051	,, Cooke, F. ..	,,
5381	,, McColl, C. S...	,,	10309	,, Connah, J. ..	,,
1928	,, Orr, A. ..	,,	10345	,, Eastwood, S...	,,
3578	,, Reilly, E. ..	,,	10514	,, Gibson, H. ..	,,
309	,, Turner, A. J.	,,	402	,, Sebeck, A. ..	,,
7778	,, Walton, T. ..	2.7.15	7441	,, Cousins, A. E.	,,
10459	,, Murphy, J. ..	,,	10339	,, Caton, J. ..	,,
2971	,, Jones, H. W...	,,	10397	,, Dawes, T. ..	,,
6105	,, Norris, S. ..	,,	10348	,, Gibbons, C. J.	,,
1756	,, Connolly, F. P.	,,	10859	,, Sibley, W. J.	,,
2025	,, Dawson, S. ..	,,	6262	,, Lovegrove, E. A. J.	15.7.15
7545	,, Heath, W. N.	,,			
3792	,, Bravery, J. W.	,,	6795	,, Norrie, C. ..	,,
4325	,, Turtle, J. ..	28.6.15	1320	,, Johnstone, W.	,,
7228	,, Whelan, J. ..	,,	6244	,, Dewey, R. J.	20.7.15

ADVANCEMENT OF PRIVATES (CORPS PAY).

The following advancements in rate of Corps Pay will take effect from July 28, 1915 :—

To be advanced to the Third Rate (at 8d.).

As Orderlies.

No.	Name	No.	Name	No.	Name
6189	Butler, W.	5709	Hammond, E.	4474	Rimmer, T.
5533	Cheeseman, G.	6535	Potter, H. H.	74	Pengelly, W.
702	Player, A. J.	6340	Jones, S. A.	19311	Cantello, H. J.
6286	Davies, P. F.	6208	Sage, J. W.	195	Corston, H. R.
265	Clarke, H. E.	16503	McKinley, A.	6492	Leggett, A. T.
6649	Alcock, H.	6469	Row, H.	17475	Lannon, P. J.
6371	Vine, C. H.	6051	Richards, W.		
12536	Bethell, B.	4332	Jacks, R. H.		

To be advanced to the Fourth Rate (at 6d.).

As Orderlies.

9596	Cooper, F.	5786	Burgess, F.	7433	Berryman, H. T.
4499	Browning, S.	7390	Orme, C.	10051	Cooke, F.
4486	Campion, B. R.	10627	Porter, H.	10309	Connah, J.
1570	Gorton, C.	400	Gallagher, P.	10514	Gibson, H.
4464	Sewell, F. J.	7529	Capel, J. W.	10339	Caton, J.
392	Sparks, J.	6971	Collier, G. E.	10397	Dawes, T.
10718	Stone, R. F.	12641	Hurst, A. H.	10348	Gibbins, C. J.
10642	Allan, H.	7799	McCord, S.	10859	Sibley, W. J.
7708	Earl, A. A.	4723	Samson, W.	18049	Bloomfield, O. G.
18652	Playle, T.	6399	Taylor, B. L.	19027	Higham, J.
6791	Fletcher, J.	16374	Barney, E. H.	456	McMillan, T.
1188	Doyle, M.	20148	Bell, E. J. S.	708	Owen, A.
19116	Dodghson, T.	6537	Bedford, F. A.	842	Parsons, S.
5302	Hayter, J. W.	20407	Bryan, T. W.	2745	Martin, C.
3109	Rice, S.	20462	Chadwick, A.	6262	Lovegrove, E. H. J.
6114	Love, G. G.	16349	Christie, G.	6785	Norrie, C.
3491	Marston, W.	17715	Claydon, G.	1320	Johnstone, W.
2555	Molloy, T.	209	Croxford, C.	10066	Clarkson, A.
28	Bracken, J.	17782	Curry, H.	19273	Groessel, G.
3232	Smyth, J.	20610	Elliott, W. A.	5199	Kitts, H. H.
10679	Upfold, L. A.	4883	Long, J. W.	7220	Shipley, F. S.
18653	Flower, F.	7328	Manning, J.	20765	Werry, T. J.
16427	Brennan, G.	6129	McDermott, J.	2779	Williams, G. H.
15907	Ashbrook, C. H.	7864	Merrill, C. C.	3904	Weale, J.
7778	Walton, T.	2845	Moore, F.	19114	Bindley, J.
10459	Murphy, J.	5519	Nicolle, W.	17494	Peckham, F.
2971	Jones, H. W.	7754	Reast, A. W.	6422	Cowie, J.
4318	Hayes, W.	10800	Simpson, H.	6663	Pickersgill, E.
6105	Norris. S.	4633	Sowden, C.	7295	Proud, E. E.
1756	Connolly. F. P.	136	Wood, C. H.	6266	Wrench, A. E.
20562	Hall, G. E.	4492	Edgar, R.	2754	Lindsay, E.
2025	Dawson, S.	10043	Loxley, E. A.	4753	Howell, R. H.
3792	Bravery, J. W.	4538	West, J. B. T.	9978	Bravington, R. J.
7831	Miller, S.	861	Matheson, A.		

As Clerks.

10694	Elliott, R. S.	7144	Cooper, E. P.	5876	Tait, W.
20360	Lowes, J. W.				

As Cooks.

6117	Higgins, A.	5580	Hawton, J. T.	5666	West, H. D. M.

The advancements are subject to the conditions laid down in paragraph 35 Standing Orders for the Royal Army Medical Corps.

Buglers.

The following boys are appointed Buglers from the dates specified :
No. 10595 Boy C. A. Ashford, June 16, 1915.
No. 12491 Boy G. M. B. Smith, June 23, 1915.

RE-POSTING TO CORPS.

The undermentioned N.C.O. rejoined the Corps on the date specified :—
No. 7564 Staff-Serjeant W. Ward, November 5, 1914, from Territorial Force.

PROMOTIONS CANCELLED.

(*a*) The promotion to the rank of Corporal notified in Corps Order No. 55 dated December 12, 1914, and to the rank of Serjeant in Corps Order No. 2 dated January 25, 1915, of No. 6392 E. A. Spurgeon is hereby cancelled as it has now been unofficially reported to the War Office that he died at the Battle of Mons.
(*b*) The promotion to the rank of Serjeant notified in Corps Order No. 51 dated June 21, 1915, of No. 18029 Corporal A. L. Burr is hereby cancelled.

ADVANCEMENT CORPS PAY CANCELLED.

(*a*) The advancement of the undermentioned Private to the 3rd rate of Corps pay as orderly, notified in Corps Order No. 54 dated June 21, 1915, is hereby cancelled : —

No. 18719 Walker, S. F.

(*b*) The advancement of the undermentioned Private to the 4th rate of Corps pay as orderly, notified in Corps Order No. 54 dated June 21, 1915, is hereby cancelled :—

No. 6965 Ness, E. R.

AMENDMENTS—CORPS ORDERS.

(*a*) In Corps Order No. 2, dated January 25, 1915, under the heading "To be Serjeant Major," the promotion of No. 12522, Quartermaster Serjeant S. Gallie, is antedated (without back pay) to August 10, 1914, with seniority next below No. 12058 Serjeant Major A. W. Pettley. (*War Office letter No.* 18/*R.A.M.C.*/786 (*A.M., D.I.*) *dated* July 24, 1915).
(*b*) In Corps Order No. 18 dated March 6, 1915, the Corps number of Lance Serjeant H. Johnson, promoted to be Serjeant, should read 19385 and not 19383.

NON-EUROPEAN SECTION, R.A.M.C., ADVANCEMENT OF PRIVATES (CORPS PAY).

The following advancements in rate of Corps Pay will take effect from July 28, 1915.

To be Advanced to the Third Rate (at 8d.).

As Orderly.

No. 15 Adams, J.

To be Advanced to the Fourth Rate (at 6d.).

As Orderly.

No. 16 Atkinstall, R. I.

NOTICE.

Cooking Section.

It is notified for general information that approval has been given for the modification, during the war, of the system of training for cooks laid down in Appendix 7 Standing Orders, R.A.M.C., on the following conditions :—

(1) Cooks in Military Hospitals.—Men who have been employed as cooks for more than three months may, if considered efficient, be examined by a Regular Officer of the unit and a Superintending Cook if available, and if they pass the examination will be eligible for registration as cooks in Military Hospitals, and for advancement to the 4th rate of Corps Pay.

(2) Superintending Cooks in Military Hospitals.—Selected men who are considered likely to be suitable for the position may be sent for instruction in accordance with Appendix 7 to a General Hospital for at least three months. For this purpose all general hospitals will be considered, for the duration of the War, as eligible to hold classes of instruction in the same manner as the hospitals referred to in paragraph 361 Standing Orders R.A.M.C.

(Authority No. 103/Miscellaneous/359 (A.M.D.I.), dated War Office July 6, 1915).

Examinations in the case of cooks in Military Hospitals may be oral and practical only, but for Superintending Cooks the procedure laid down in paragraph 379-384 Standing Orders, R.A.M.C., should be followed.

TERRITORIAL FORCE.

1*st London* (*City of London*) *General Hospital*—The undermentioned Lieutenants to be Captains, dated April 1, 1915: Alfred B. P. Smith; Lionel G. Crossman, M.B.; Martin W. K. Bird; Arnold W. Slott, and to remain seconded.

1*st London General Hospital.*—The date of the appointment of Lieutenant John Duncan Legge Currie is June 17, 1915, and not as stated in the *London Gazette* of August 7, 1915.

1*st London* (*City of London*) *Sanitary Company.*—John Oglethorpe Wakelin Barrett, M.B., to be Lieutenant, dated June 25, 1915; Major Greenwood to be Lieutenant, dated August 9, 1915; Percival Hartley to be Lieutenant, dated August 10, 1915. The date of appointment of Lieutenant Norman A. Dore is June 29, 1915, and not as stated in the *London Gazette* of August 5, 1915. The date of appointment of Lieutenant Horace G. Moss is June 29, 1915, and not as stated in the *London Gazette* of August 5, 1915. Lieutenant James E. Wilson, M.D., from the 3rd North Midland Field Ambulance, to be Lieutenant, dated August 22, 1915. Cadet George Leslie Matthews, from the University of London Contingent, Senior Division, Officers Training Corps, to be Lieutenant, dated August 28, 1915. Ernest Romney Matthews to be Lieutenant, dated August 28, 1915.

2*nd London* (*City of London*) *General Hospital.*—The undermentioned Lieutenants to be Captains: Cyril E. Petley, dated April 1, 1915; Leonard Milton, dated April 1, 1915; Herbert Sharpe, dated April 1, 1915; Kenneth B. Clarke, dated August 1, 1915.

2*nd London* (*City of London*) *Field Ambulance.*—Transport Officer and Honorary Lieutenant Arthur D. Don resigns his commission, dated August 26, 1915; Lieutenant Charles Eustace Williams, from the 3rd London (City of London) Field Ambulance to be Lieutenant, dated August 28, 1915.

2*nd London Sanitary Company.*—Serjeant Thomas Jenkins Murray, from 3rd North Midland Field Ambulance, to be Lieutenant, dated August 7,1915.

3*rd London General Hospital.*—The undermentioned Lieutenants to be Captains and to remain seconded, dated April 1, 1915: George H. D. Webb, Edward Smeed, Walter Henry Lloyd to be Lieutenant, dated August 12, 1915. The undermentioned Lieutenants are seconded for duty with the 1st Highland Field Ambulance, dated August 31, 1915: John St. A. Titmas, Harry A. Lucas, John B. Rawlins, Philip W. Green.

4*th London General Hospital.*—Major William A. Turner, M.D., is seconded, dated May 26, 1915.

4*th London Field Ambulance.*—William Morgan Langdon to be Lieutenant, dated August 28, 1915. Edgar Alfred Lambert to be Transport Officer, with the honorary rank of Lieutenant, dated July 28, 1915.

London Mounted Brigade Field Ambulance.—Captain Hugh S. Beadles to be temporary Major, dated June 1, 1915.

London Sanitary Company.—Lieutenant Vincent P. Norman to be Captain, dated August 5, 1915.

Scottish Horse Mounted Brigade Field Ambulance.—Cadet Francis Murray Halley, from the Edinburgh University Contingent, Senior Division, Officers Training Corps, to be Lieutenant, dated July 10, 1915.

2*nd Scottish General Hospital.*—The undermentioned officers are seconded, dated September 10, 1915: Lieutenant-Colonel Charles W. Cathcart, M.B., F.R.C.S.; Lieutenant-Colonel Sir Robert W. Philip, M.D.; Major David Wallace, C.M.G., M.B.,

F.R.C.S. ; Major Francis D. Boyd, C.M.G., M.D. ; Captain Alexander Miles, M.D., F.R.C.S. ; Captain John W. Dowden, M.D., F.R.C.S. ; Captain Archibald A. S. Skirving, C.M.G., M.B., F.R.C.S. ; Captain James G. Cattanach, M.B. ; Captain John Eason, M.D. ; Captain W. Simpson, M.D. ; Captain Edwin Matthew, M.B.

4th Scottish General Hospital.—Major Duncan O. MacGregor, M.B., resigns his commission on account of ill-health, dated June 30, 1915.

Highland Mounted Brigade Field Ambulance.—The undermentioned Lieutenants to be Captains, dated April 1, 1915 : George G. Middleton, M.B. ; Evan A. Mackenzie ; Thomas E. Roberts, M.D. ; Transport Officer and Honorary Lieutenant Charles Riggall resigns his commission, dated August 24, 1915.

1st Highland Field Ambulance.—Captain Arthur Kellas, M.B., to be temporary Major, dated January 14, 1915 ; Lieutenant William H. E. Brand to be Captain, dated April 7, 1915 ; Major James Robertson, M.D., to be temporary Lieutenant-Colonel, dated May 1, 1915 ; Alfred Bell Whitton, M.B. (late Lieutenant Colonel, 6th Battalion, Gordon Highlanders), to be Captain (temporary), dated June 25, 1915 ; Captain George Dick, M.B., from the Sanitary Service, to be Captain, dated September 11, 1915.

2nd Highland Field Ambulance.—Lieutenant Thomas S. Slessor, M.B., to be Captain, dated June 18, 1915. The undermentioned to be Lieutenants, dated August 12, 1915 : Alexander Main Baillie ; Harry Gordon Donald, John William McKeggie ; Cadet James Alexander Seller, from the Aberdeen University Contingent, Senior Division, Officers Training Corps ; William Charles Davidson Wilson.

3rd Highland Field Ambulance.—The date of appointment of Lieutenant Alexander B. Jamieson, M.B., is June 1, 1915, and not as stated in the *London Gazette* of June 7, 1915.

Lowland Mounted Brigade Field Ambulance.—The undermentioned Lieutenants to be Captains : Andrew R. Muir, M.B., dated April 1, 1914 ; Hugh Forest, M.B., dated April 1, 1915 ; Andrew M. Young, M.B., dated May 4, 1915 : Edward N. Thomson, M.B., dated June 11, 1915.

Lowland Field Ambulance.—Lieutenant Robert A. Lennie, M.B., to be Captain, dated June 30, 1915 ; Lieutenant Colin C. Philip, M.B., to be Captain, dated July 13, 1915 ; the undermentioned Lieutenants to be Captains, dated April 1, 1915 : James R. Menzies, M.B. ; William H. Armistead, M.B.

1st Lowland Field Ambulance.—Ernest Switzer Forde (late Captain, 5th (Dumfries and Galloway) Battalion, The King's Own Scottish Borderers, to be Major (temporary), dated July 16, 1915 ; James Stark Linton to be Quartermaster, with the honorary rank of Lieutenant, dated August 28, 1915.

2nd Lowland Field Ambulance.—Harry Taylor Findlay, M.B., to be Lieutenant, dated August 10, 1915.

3rd Lowland Field Ambulance.—The undermentioned Lieutenants to be Captains, dated April 1, 1915 : Robert B. Barnetson, M.B. ; James A. Henderson, M.B.

1st Northumbrian Field Ambulance.—Major Edward Turton, M.D., to be temporary Lieutenant-Colonel, dated September 11, 1915.

3rd Northumbrian Field Ambulance.—Patrick Joseph Sheedy to be Lieutenant, dated July 31, 1915 ; Captain Francis R. Eddison, from Attached to Units other than Medical Units, to be Captain, dated September 11, 1915 ; John Austin Lee Magee (late Second Lieutenant, 10th (Service) Battalion, The Durham Light Infantry), to be Lieutenant, dated September 11, 1915.

West Riding Casualty Clearing Station.—The undermentioned Lieutenants to be Captains : Robert G. Dixon, M.B., dated April 1, 1915 ; Hubert W. Symons, M.B., dated April 1, 1915 ; Frederick P. Gibson, M.B., dated April 23, 1915 ; Hubert T. Bates, M.B., dated April 23, 1915 ; Jeffrey W. Malim, M.B., dated April 23, 1915 ; David R. Cramb, M.B., dated April 23, 1915 ; George P. P. Clapham, dated April 23, 1915 ; Arthur S. Hebblethwaite, M.B., dated June 30, 1915 ; Charles M. Gozney, M.B., dated July 1, 1915

1st West Riding Field Ambulance.—Lieutenant Edwin S. G. Fowler to be Captain, dated June 25, 1915.

2nd West Riding Field Ambulance.—Lieutenant Craufurd T. Matthews, M.B., to be Captain, dated April 1, 1915 ; Lieutenant Digby Wrangham Hardy, M.B., to be Captain, dated June 27, 1915.

3rd West Riding Field Ambulance.—Captain William J. Gray, from Attached to Units other than Medical Units, to be Captain, dated August 25, 1915.

Yorkshire Mounted Brigade Field Ambulance.—Lieutenant William H. N. White, M.B., to be Captain, dated May 2, 1915.

2nd East Lancashire Field Ambulance.—Ernest Lionel Forward to be Lieutenant, dated June 30, 1915; Arthur William Havard, M.B., to be Lieutenant, dated June 30, 1915.

1st West Lancashire Field Ambulance.—Captain Richard S. Taylor, M.B., F.R.C.S., to be temporary Major, dated May 29, 1915; the following announcement is substituted for that which appeared in the *London Gazette* of July 19, 1915: Captain William H. Broad, M.D., 6th (Rifle) Battalion, The King's (Liverpool Regiment), to be Captain (temporary), dated May 26, 1915.

2nd West Lancashire Field Ambulance.—Quartermaster-Serjeant William Holden Emblem, from the 3rd West Lancashire Field Ambulance, to be Quartermaster, with the honorary rank of Lieutenant, dated September 11, 1915.

Notts and Derby Mounted Brigade Field Ambulance.—The undermentioned Lieutenants to be Captains: Lewis Moysey, M.B., dated April 22, 1915; William A. Brechin, M.B., dated June 16, 1915; Frank Standish, dated June 25, 1915; the appointment of John Wootton Rammell as Lieutenant bears date June 21, 1915, and not as stated in the *London Gazette* of July 20, 1915; David William Griffiths to be Lieutenant, dated July 27, 1915.

1st North Midland Field Ambulance.—The undermentioned Lieutenants to be Captains: Hugh A. Macmillan, M.B., dated April 20, 1915; Matthew T. Ascough, dated April 22, 1915; Thomas S. Elliot, dated April 28, 1915; Alexander Fordyce, M.B., dated May 7, 1915; Arthur Heath, M.D., F.R.C.S., dated May 24, 1915.

2nd North Midland Field Ambulance.—The undermentioned Lieutenants to be Captains: Henry P. Malcolm, M.B., dated May 10, 1915; Robert J. McConnell, M.B., dated May 10, 1915; Edward C. T. Emerson, M.B., dated May 19, 1915; Joshua Buchanan McLean, M.B., to be Lieutenant, dated August 19, 1915.

3rd North Midland Field Ambulance.—The undermentioned Lieutenants to be Captains: Charles C. Grummit, dated April 12, 1915; John G. J. Green, dated April 19, 1915; James E. S. Smith, M.B., dated July 22, 1915; George F. Denning, dated July 23, 1915; Bertram M. Footner, from Attached to Units other than Medical Units, to be Captain, dated August 24, 1915.

North Midland Mounted Brigade Field Ambulance.—The undermentioned Lieutenants to be Captains: John B. Stanley, dated April 1, 1915; Samuel Acheson, M.B., dated April 26, 1915: Sydney Alan Stormer Malkin to be Lieutenant, dated August 5, 1915.

North Midland Casualty Clearing Station.—Albert Richard Henchley, M.D. (late Major, 2nd Home Counties Field Ambulance), to be Major (temporary), dated July 14, 1915.

1st South Midland Mounted Brigade Field Ambulance.—Lieutenant James H. Wilkinson to be Captain, dated April 19, 1915; Lieutenant George C. Soutter, M.D., to be Captain, dated May 3, 1915. The following announcement is substituted for that which appeared in the *London Gazette* of July 12, 1915:—Captain Frederic E. France, M.B., from Attached to Units other than Medical Units, to be Captain, dated May 27, 1915; Major Thomas Henderson Forrest, M.B., to be Lieutenant-Colonel, dated August 11, 1915.

2nd South Midland Mounted Brigade Field Ambulance.—Lieutenant Alfred G. Levy, M.B., to be Captain, dated August 1, 1915; Lieutenant Alexander Rodger, M.B., to be Captain, dated August 14, 1915; Lieutenant John C. S. Dunn to be Captain, dated August 27, 1915.

3rd South Midland Field Ambulance.—Lieutenant Frank T. Boucher to be Captain, dated April 1, 1915; William George McKenzie to be Lieutenant, dated August 14, 1915; Henry Neville Crowe, M.D., to be Lieutenant, dated September 11, 1915.

Welsh Field Ambulance.—Captain Henry J. Dunbar, M.B., to be temporary Major, dated April 30, 1915; Lawrence Stephenson to be Quartermaster, with the honorary rank of Lieutenant, dated July 25, 1915.

Welsh Border Mounted Brigade Field Ambulance.—Haldane Carson Gilmore to be Lieutenant, dated July 11, 1915.

1*st Welsh Field Ambulance.*—John Cook (late Second Lieutenant, 4th Welsh Brigade, Royal Field Artillery) to be Captain (temporary), dated July 2, 1915.

South Wales Mounted Brigade Field Ambulance.—The undermentioned Lieutenants to be Captains: Archibald J. Campbell, dated April 1, 1915; Ernest L. Sandiland, M.B., dated April 1, 1915; Martin Scales, dated April 21, 1915; Lieutenant John A. Cooke to be Captain, dated June 26, 1915.

1*st East Anglian Field Ambulance.*—Lieutenant Arthur W. Paterson, M.B., to be Captain, dated May 29, 1915.

2*nd East Anglian Field Ambulance.*—The surname of Lieutenant Arthur Greene, M.D., F.R.C.S., is as now stated, and not as announced in the *London Gazette* of July 20, 1915.

3*rd East Anglian Field Ambulance.*—Lieutenant William R. M. Turtle, M.B., to Captain, dated April 1, 1915; Lieutenant Sigismund H. Rentzsch to be Captain, dated June 24, 1915; Major Henry A. Ridyard, from Attached to Units other than Medical Units, to be Major, dated July 10, 1915; Major Henry A. Rudyard to be temporary Lieutenant-Colonel, dated July 10, 1915.

1*st Northern General Hospital.*—The undermentioned Lieutenants to be Captains, dated August 9, 1915: Frederick J. Nattrass, M.B., Sydney Thompson, M.B.

2*nd Northern General Hospital.*—Ethelbert Rest Flint to be Lieutenant, dated August 10, 1915.

4*th Northern General Hospital.*—Lieutenant-Colonel George H. Grimoldby resigns his commission on account of ill-health, dated September 11, 1915.

1*st Southern General Hospital.*—George Joughin, M.B., to be Lieutenant, dated July 21, 1915; Koch Harry Gill, M.B., to be Lieutenant, dated July 21, 1915; Geoffrey Legh Wilkinson to be Lieutenant, dated September 3, 1915.

2*nd Southern General Hospital.*—Major Arthur B. Prowse, M.D., F.R.C.S., to be temporary Lieutenant-Colonel, dated July 10, 1915; Lieutenant Robert J. Irving, M.B., F.R.C.S., to be Captain, dated July 19, 1915.

Eastern General Hospital.—The undermentioned Lieutenants to be Captains, dated April 1, 1915; John H. Owens, Ffrangcon Roberts.

1*st Eastern General Hospital.*—Serjeant-Major Charles Edward Wilkin Coe to be Quartermaster, with the honorary rank of Lieutenant, dated August 14, 1915.

Wessex Divisional Sanitary Section.—Arthur Ernest Bonham to be Lieutenant, dated August 12, 1915.

1*st Wessex Field Ambulance.*—Lieutenant Frank A. Roper, M.B., to be Captain, dated April 1, 1915; Lieutenant Alfred J. H. Iles to be Captain, dated April 1, 1915; Lieutenant George D. Perry resigns his commission on account of ill-health, dated September 11, 1915.

2*nd Wessex Field Ambulance.*—Lieutenant Angus Cameron, M.B., from Attached to Units other than Medical Units, to be Lieutenant, dated May 8, 1915; Major Harry C. Parsons to be temporary Lieutenant-Colonel, dated August 25, 1915.

South Western Mounted Brigade Field Ambulance.—Lieutenant Philip W. Mason, M.B., to be Captain, dated April 3, 1915: Lieutenant Harold Burnet Porteous, M.B., to be Captain, dated April 2, 1915; Lieutenant Bertram Michell Young, to be Captain, dated April 16, 1915.

1*st South Western Mounted Brigade Field Ambulance.*—Hugh Wansey Bayly (late temporary Surgeon, Royal Navy) to be Captain (temporary), dated September 4, 1915.

2*nd South Western Mounted Brigade Field Ambulance.*—Lieutenant Louis J. E. McHugh, M.B., to be Captain, dated April 6, 1915.

1*st Western General Hospital.*—The seconding of Captain Keith W. Monsarrat, M.B., which was announced in the *London Gazette* of May 20, 1915, is cancelled; Major Claude Rundle, M.D., to be temporary Lieutenant-Colonel, dated August 11, 1915.

2*nd Western General Hospital.*—Graham Miles Benton, M.B., to be Captain, whose services will be available on mobilization, dated June 14, 1915; James Gladstone McKinlay, M.B., to be Lieutenant, dated June 14, 1915; Edward Moir to be Captain, whose services will be available on mobilization, dated August 1, 1915.

3*rd Western General Hospital.*—Rhys Trevor Jones to be Lieutenant, dated June 16, 1915; Cornelius Charles Boyle, M.B., to be Lieutenant, dated June 20, 1915; John Thomas Williams, M.D., to be Lieutenant, dated June 30, 1915; Serjeant-Major Charles Hayward to be Quartermaster, with the honorary rank of Lieutenant, dated August 24, 1915.

3rd Home Counties Field Ambulance.—Captain Augustine Griffith, M.D., from the Sanitary Service, to be Captain, dated August 31, 1915; Corporal Stanley Stephens Eyre, from the West Kent (Queen's Own) Yeomanry, to be Transport Officer, with the honorary rank of Lieutenant, dated September 7, 1915; Major Hector G. G. Mackenzie, M.D., to be temporary Lieutenant-Colonel, dated September 11, 1915; Captain William D. Sturrock, M.D., to be temporary Major, dated September 11, 1915.

Home Counties Casualty Clearing Station.—William Maskelyne Parham, M.D. (late Surgeon-Captain, 1st Volunteer Battalion, Princess Charlotte of Wales's (Royal Berkshire Regiment), to be Captain, dated September 4, 1915.

ATTACHED TO UNITS OTHER THAN MEDICAL UNITS.

Lieutenant Harry M. Soden, from the 3rd Welsh Field Ambulance, to be Lieutenant, dated December 15, 1914.

Major Frederick J. Oxley, from the 17th Battalion, the London Regiment, to be Major, dated September 2, 1915.

Lieutenant George B. Forge to be Captain, dated April 1, 1915.
Lieutenant John Saffley, M.B., to be Captain, dated April 1, 1915.
Lieutenant Francis R. Eddison to be Captain, dated April 1, 1915.
Lieutenant Stephen O. Dolan to be Captain, dated April 1, 1915.
Lieutenant Ernest E. B. Landon to be Captain, dated April 1, 1915.
Lieutenant Charles E. Anderson to be Captain, dated April 1, 1915.
Lieutenant Reginald C. Neil to be Captain. dated April 1, 1915.
Lieutenant Charles J. Fox to be Captain, dated April 1, 1915.
Lieutenant Hastings F. Everett to be Captain, dated April 1, 1915.
Lieutenant Jesse R. Garrood, M.D., to be Captain, dated April 1, 1915.
Lieutenant George Candler to be Captain, dated April 5, 1915.
Arthur Benjamin Winder, M.D., to be Lieutenant, dated June 1, 1915.
Lieutenant William Love to be Captain, dated April 12, 1915.
Lieutenant Edward L. Martin, M.D., to be Captain, dated April 15, 1915.
Lieutenant John J. Weaver to be Captain, dated April 15, 1915.
Lieutenant Frederick E. France, M.B., to be Captain, dated April 27, 1915.
Lieutenant Austen A. Bearne to be Captain, dated May 1, 1915.
Lieutenant Walter Smartt, F.R.C.S.I., to be Captain, dated August 11, 1915.
Leonard Stephen Willox, M.D., to be Lieutenant, dated August 5, 1915.
Lieutenant Samuel Rutherford, M.B., to be Captain, dated June 10, 1915.
Lieutenant Hugh G. Bruce, M.B., to be Captain, dated July 24, 1915.
Lieutenant James McGavin Deuchars, M.B., to be Captain, dated August 4, 1915.
Captain Charles A. C. Smelt, M.B., to be Major, dated May 27, 1915.
Surgeon-Major John Nightingale, M.D., from the 1st West Riding Brigade, Royal Field Artillery, to be Major, dated September 7, 1915.
Lieutenant Frederick Hunton, M.D., to be Captain, dated June 17, 1915.
Frederick Arthur Pring to be Lieutenant, dated August 19, 1915.
Wilfred Percy Tindal Atkinson to be Lieutenant, dated August 20, 1915.
Captain Owen W. D. Stell, from the 3rd Battalion, The Monmouthshire Regiment, to be Captain, dated September 10, 1915.
Lieutenant Hugh A. McLean, M.B., to be Captain, dated June 6, 1915.
Captain Thomas D. Laird, M.B., to be Major, dated August 1, 1915.
Captain Thomas Douglas Brown, M.B., to be Major, dated August 5, 1915.
Lieutenant Sydney Oliphant Bingham, from the 14th (Reserve) Battalion, The Sherwood Foresters (Nottinghamshire and Derbyshire Regiment), to be Lieutenant, dated August 22, 1915.

The undermentioned Lieutenants to be Captains :—
Alexander Ambrose, M.D., dated April 1, 1915.
Ernest S. Stork, M.B., dated April 1, 1915.
William A. Robertson, dated April 1, 1915.
Alexander G. V. Van Someren, M.B., dated April 1, 1915.
Maurice C. Anderson, dated April 1, 1915.
Reginald D. Gawn, M.B., dated April 1, 1915.
John C. McKenzie, M.B., dated April 1, 1915.
George H. Dominy, dated April 9, 1915.
John A. Thomson, M.B., dated April 24, 1915.
Archibald N. S. Carmichael, M.B., dated April 27, 1915.
Ernest B. Keen, dated May 1, 1915.
Harold F. L. Hugo, M.B., dated June 19, 1915.

John W. M. Jamieson, M.B., dated June 21, 1915.
Thomas Rhind, dated June 28, 1915.
William H. Date, M.D., dated August 6, 1915.
Captain Thomas E. Roberts, M.D., from the Highland Mounted Brigade Field Ambulance, to be Captain, dated July 26, 1915.
Lieutenant George B. H. Jones, M.D., to be Captain, dated April 1, 1915.
Lieutenant William E. L. Elliott, M.D., to be Captain, dated April 1, 1915.
Lieutenant Charles A. Sampson to be Captain, dated May 16, 1915.
Lieutenant Robert L. Guthrie, M.D., to be Captain, dated June 15, 1915.
Lieutenant Joseph B. McKay resigns his commission on account of ill-health, dated August 13, 1915.
Captain Owen W. D. Steel, from the 3rd Battalion, the Monmouthshire Regiment, to be Captain, dated September 10, 1915.
The seconding of Captain Haldinstein D. Davis, which was announced in the *London Gazette* of November 13, 1914, is cancelled.

TERRITORIAL FORCE RESERVE.

Royal Army Medical Corps.

Cadet Alexander Louis Cameron Mackenzie, from the Aberdeen University Contingent, Officers Training Corps, to be Lieutenant (on probation), dated August 20, 1915.
Ex-Cadet John Taylor Scrogie, M.B., from the Aberdeen University Officers Training Corps to be Lieutenant (on probation), dated July 31, 1915.
Ex-Cadet Alan Rupert Laurie, M.D., from the Edinburgh University Contingent, Officers Training Corps, to be Lieutenant (on probation), dated August 1, 1915.
Captain Owen Wilson, M.B., resigns his commission, dated July 16, 1915.
Temporary Lieutenant Kenneth Alexander Maclean, M.B., from Royal Army Medical Corps, to be Captain, dated August 16, 1915.
The undermentioned Lieutenants to be Captains :—
William J. Reid, M.B., dated April 1, 1915.
Ronald W. Duncan, dated July 10, 1915.
William L. E. Reynolds, dated August 1, 1915.
Frank C. Harrison, dated August 3, 1915.
William K. Campbell, M.B., dated August 6, 1915.
Charles M. G. Campbell, dated August 8, 1915.
Owen G. Parry Jones, dated August 8, 1915.
Eris S. Mawe, dated August 9, 1915.
William B. Jepson, dated August 12, 1915.
Edward W. Mann, M.B., dated August 13, 1915.
Thomas D. Inch, M.B., dated August 15, 1915.
Gerald F. V. Leary, M.B., dated August 16, 1915.
Sidney W. Lund, M.B., dated August 16, 1915.
Noel H. W. Saw, dated August 20, 1915.
Henry R. Sheppard, dated August 22, 1915.
Donald C. Macdonald, M.B., dated August 22, 1915.
William T. Hare, dated August 24, 1915.
Herbert Troughton Chatfield, M.B., to be Lieutenant (on probation), dated August 20, 1915.
The undermentioned Lieutenants (on probation) are confirmed in their rank :—
Douglas C. Pim, M.B.
Norman L. Reis, M.B.
William J. Dowling, M.B.
Frederick R. S. Shaw, M.B.
Cyril E. H. Gater.
Charles H. Brennan.
Samuel D. Lodge.
Arthur J. Beveridge, M.B.
John G. Hendry, M.B.
Ribton G. Blair, M.B.
George Stanton, M.B.
John D. Proud, M.B.
John A. Musgrave.
Charles M. G. Campbell.
James McKay, M.B.

DEATHS.

JONES.—On August 30, at Bryn Tirion, Church Walks, Llandudno, Colonel John Matthew Jones, A.M.S., late A.D.M.S. Plymouth District. Invalided whilst on active service.

FLEMING.—Brigade Surgeon Lieutenant-Colonel Joseph Fleming, M.D., F.R.C.S.Edin., retired, Army Medical Staff, died at Castlequarter, Inch, Londonderry, on August 10, 1915, aged 75.

LAMPREY.—Lieutenant-Colonel Joseph John Lamprey, retired, Army Medical Staff, died at 1, Schubert Road, Southfields, London, S.W., on September 6, 1915, aged 66.

EXCHANGES, &c.

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Notices.

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The Editor will be glad to receive original communications upon professional subjects, travel, and personal experiences, etc. He will also be glad to receive items of news and information regarding matters of interest to the Corps from the various garrisons, districts, and commands at home and abroad.

All such Communications or Articles accepted and published in the "Journal of the Royal Army Medical Corps" will (unless the Author notifies at the time of submission that he reserves the copyright of the Article to himself) become the property of the Library and Journal Committee, who will exercise full copyright powers concerning such Articles.

Matter intended for the Corps News should reach the Editor not later than the 15th of each month for the following month's issue. Notices of Births, Marriages, and Deaths are inserted free of charge to subscribers and members of the Corps. All these communications should be written upon one side of the paper only; they should by preference be type-written, but, if not, all proper names should be written in capital letters (or printed) to avoid mistakes, and be addressed The Editor, "JOURNAL OF THE ROYAL ARMY MEDICAL CORPS," War Office, Whitehall, London, S.W.

Communications have been received from Sir G. Makins, Colonel W. W. Pike, D.S.O., Lieutenant-Colonel F. C. Heffernan, Major F. W. Cotton, P. Sargent, Major F. A. H. Clarke, G. Holmes, Captain J. Oliver Hamilton, Lieutenant C. Clarke, James Rae, Lieutenant J. S. Goodacre.

The following publications have been received:—

British: Tropical Diseases Bulletin, Medical Press and Circular, The Journal of Tropical Medicine and Hygiene, The Lancet, The Hospital, The Indian Medical Gazette, Journal of the Royal United Service Institution, The Journal of State Medicine, The Medical Journal of South Africa, The Royal Engineers' Journal, St. Bartholomew's Hospital Journal, Medical Journal of Australia, The Sanitary Record and Municipal Engineering, The Medical Review, The Practitioner, Red Cross and Ambulance News, The Army Service Corps Journal, Australian Military Journal, Guy's Hospital Gazette, The Indian Journal of Medical Research.

Foreign: Annali di Medicina Navale e Coloniale, Russian Naval Medical Journal, Bulletin de l'Institut Pasteur, Bulletin of the Johns Hopkins Hospital, Revista de Sanidâd Militar, United States Naval Medical Bulletin, The Military Surgeon, Le Caducée.

MANAGER'S NOTICES.

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